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TUBERCULIN
IN THE
DIAGNOSIS AND TREATMENT
OF TUBERCULOSIS

TUBERCULIN

IN THE

DIAGNOSIS AND TREATMENT OF TUBERCULOSIS

(WEBER-PARKES PRIZE ESSAY 1909)

WITH ADDITIONS

BY

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LONDON

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1912

DEDICATED
BY PERMISSION
TO HIS EXCELLENCY
PROFESSOR ROBERT KOCH,
WHO, THOUGH DEAD, STILL LIVES
AS THE INSPIRING GENIUS
OF A FAITHFUL PUPIL'S
HUMBLE WORK.

APOLOGIA

WHOSOEVER undertakes to prove that everyone in his own particular society has been wrong, and he alone has arrived at the conscious possession of truth, takes great risks, and the odds are manifestly against him. Yet such a man may happen to be quite right. The glorious path of human progress has ever been illumined by such beacon lights. In modern times, Galileo, Harvey, Newton, Pasteur, and Koch are among these giants of the human race. Their work by its irresistible truth must survive them, and can never be undone. During their life they have been buffeted and persecuted, reviled and ridiculed, and too often have died before even their friends discovered they were right. For generations their enemies have scoffed and sneered. Nevertheless, Heaven has ordained that these discoverers of truth should have some disciples. Since 1884, when I had the honour of being a pupil of Koch, my allegiance has not once wavered, and in publishing this small work, I am compelled by a sense of duty to acknowledge that this great genius has been the source and inspiration of my life's work.

Looking at work with all its trials, its hardships, and its disappointments as part of the religious duty of man, it is encouraging and stimulating to think that one's own work may bring within reach of the poorest human creature the solace and comfort that must otherwise be denied him.

Through the profound genius of Koch, and Koch alone, a certain, safe, and trustworthy remedy has been found for the treatment of pulmonary tuberculosis, which is but the name for consumption in its first beginnings, and it is high time that this great truth should be acknowledged and inspire the whole of the medical profession to imitate the labours of those

who in season and out of season have preached this great gospel of mercy and beneficence.

To all my colleagues in this profession, in every part of the globe, I would say in all humility and earnestness, "Go thou and do likewise."

"THE PROPHETS PROPHECY FALSELY."

History tells us that pioneers in the path of progress have found it no easy task to convince the world of truths which are in advance of, and may be entirely fatal to, the opinion of the day. The scholastic philosophers of the Middle Ages insisted that the world was flat, even when the Arabs were teaching their children geography upon globes. Galileo, Newton, Harvey, Sydenham, Koch, and our own Lister were ridiculed and abused because they discerned truth where others saw nothing but error or fanaticism. In these days, at any rate, of scientific research and high culture, the discoverer of a great truth should at least have a fair chance of expounding his views, and even in difficult problems there should be always a readiness to search for truth and to prove or disprove the value of a new idea, not by mere assumptions or statements, but by direct and relevant evidence. As a disciple of Koch, I can hardly remember ever gaining a fair and impartial hearing for a mature and deliberate opinion upon the value of tuberculin in diagnosis and treatment, even though the opinion was based upon the careful analysis of prolonged and laborious investigations.

In 1901 and 1907 when I brought before the Australasian Congress my investigations upon tuberculin at the Inter-Colonial Congress in Australia, I was not allowed to finish my paper. For fully fifteen years my views, which were largely based upon the monumental labours of Koch, were received with scorn, contempt, and even ridicule. Nevertheless, I continued to work in my own way, and every year found me more loyal to and more convinced of the truth of Koch's epoch-making discoveries. I have been in England scarcely two years, and in that short space of time have learnt to my sorrow that the medical mind manifests an

extraordinary complacency towards views that owe nothing to the inspiration of modern science, and, on the other hand, fosters a strange intolerance of views which have been evolved from investigations carried out in accordance with the strictest canons of logic.

A fair illustration of this curious attitude of the medical mind may be found in a recent Report upon sanatorium treatment by such distinguished members of the medical profession as Sir Clifford Allbutt, Sir Lauder Brunton, Sir William Osler, and Dr. Arthur Latham. This report purports to be an impartial judgment upon the value of sanatorium methods, and is designed to give a definite direction to the Welsh Memorial scheme, for which, through the energy and generosity of Mr. David Davies, M.P., a magnificent sum of money has been collected for the purpose of dealing with the tuberculosis problem in Wales. This scheme, initiated as the Welsh Memorial of the beneficent reign of King Edward VII, has not yet crystallised into a definite and permanent form. It has been proposed that the erection and maintenance of sanatoria should form the central and essential feature of the undertaking, and some encouragement to this method of dealing with tuberculosis in Wales may be found in the report issued and signed by these representative men. The encouragement is certainly half-hearted and faltering; but after reading the report, one receives the impression that sanatoria may still be trusted to help even the poor who suffer from consumption.

The authors of this report carefully avoid touching upon the financial aspect of their proposals. In spite of German experiences, and of the damaging evidence to be found in the late Dr. Bulstrode's report to the Local Government Board, these authors still pin their faith to sanatoria, although their proposals are worm-eaten with utterly bad finance.

My object in traversing this discursive and disjointed report is to call attention to the extraordinary and inexplicable attitude of these authors towards the system of tuberculin dispensaries, which I have already proved to be the effective means of bringing help and relief to the vast majority of the poor who suffer from consumption. I have

demonstrated in public before scores of medical men that tuberculin dispensaries are not merely the best, but the only means of treating the vast majority of cases of pulmonary tuberculosis among the poor.

I have been working for fifteen months and more at my tuberculin dispensary in Kennington Road and during that time have dealt with almost every form of tuberculosis in every stage of the disease. My dispensary is open to the medical profession, and I can say that many scores of medical men have availed themselves of my invitation to come and see for themselves what is being done. Even at Oxford I gave a lecture and demonstration at the Radcliffe Infirmary. Professor Osler, as he then was, sent an excuse for his absence. I do not know Dr. Arthur Latham, nor do I know Sir Clifford Allbutt, or Sir Lauder Brunton, even by sight; thus these four authorities can hardly know what I am doing at my dispensary. If they know anything, it must be by hearsay, and I need not comment upon the value of hearsay evidence or vicarious experience. Nevertheless, these four authorities have signed their names to the following statements: "The success of the so-called tuberculin dispensaries rests largely on the fact that Class I. and Class II. are chiefly treated at them. A further part of their success is due to the many instances in which the diagnosis of the presence of active disease is faulty, with the result that many persons are treated with tuberculin and labelled as cures in whom the disease has previously been arrested."

There is no doubt that this is a deliberate stab in the back for me, because no other medical man is definitely associated with tuberculin dispensaries. That these medical gentlemen should conspire to deal this blow at my skill in diagnosis, although not one of them has ever witnessed me examine a case, let alone "many cures," may be within the rules of medical ethics, but is hardly consistent with the highest sense of justice the human mind can know.

I cannot feel anger, only sorrow, that four distinguished members of the medical profession should descend to accuse an ordinary member of their profession of want of skill in diagnosis, when they have themselves no means of forming

any valid or fair opinion. Their offence is all the greater because they scrupulously avoid mentioning my name. I have not been very long in London, but I have found that the medical men who have actually watched my work totally dissent from this unfair and entirely personal line of argument.

I shall now proceed to deal with the salient features of this Report, in the hope that I may still have influence enough to divert this splendid sum of money collected in Wales to a method of dealing with tuberculosis among the poor, which will easily survive the perverse and pointless criticism directed against the "so-called tuberculin dispensaries."

This report upon the value of sanatorium treatment confesses to the failure of the system, and yet urges its continuance. "In many cases it must be useless." "In a few instances it is actually harmful," and "in many cases this method of treatment need not be carried out at an institution," or, in other words, in many cases, sanatorium treatment is unnecessary. Why, then, speak of it as sanatorium treatment? Will these authorities give some definite estimate of the number of cases of consumption among the poor that may and should be treated in sanatoria? It is casting dust in the eyes of the people to vaunt the sanatorium system in one breath and then speak of sanatoria as an unnecessary luxury.

We learn from this report that "the cardinal factor in the treatment is the constant and skilled regulation of the amount of rest and exercise prescribed from day to day for each individual patient." This system is Bechmer's, as anyone who has visited Goerbersdorf very well knows, but it is dignified nowadays by the term "auto-inoculation." Certainly if this cumbersome and complicated system of "auto-inoculation" is able to do all that is required for the sufferers from consumption, these authorities are right in recommending the system to their *well-to-do* patients; but so far this old system under a new name has been applied chiefly to early and selected cases, which have already been through a probationary period, and sufficient time has not elapsed for

us to be able to say whether these results will last. It is, therefore, somewhat premature to speak of the "cardinal factor," when the results of this system in England are still *sub judice*. As yet, there is no trustworthy evidence that this system of "auto-inoculation" by graduated exercise can do more than the sanatorium methods of Germany have already achieved. Certainly such a system of "auto-inoculation" carried out under the Insurance Bill requires sanatoria.

As these authorities choose to furnish no evidence in favour of the system they advocate, and trust the public will place a high valuation upon their mere statements, I venture to furnish evidence that proves the hollowness of these pretensions that sanatoria combined with graduated exercises can help the poor suffering from consumption. Let me tell these medical men that both rest and graduated exercise have long been exploited in Germany in the treatment of consumption at sanatoria, and the results of sanatorium treatment among the poor in Germany lend no encouragement to the view that sanatoria can help the poor.

I give at pages 215-217 the experiences of Moeller, Weicker, and others, who carefully tried the system of sanatorium treatment.

The tragic results of sanatorium treatment compelled both Moeller and Weicker to look about for some better system, and they are both among the most enthusiastic advocates of tuberculin treatment in Germany. Weicker's investigations are invaluable as evidence upon this national question, because he was dealing with the very class of people who would come within the scope of Mr. Lloyd George's Insurance Bill.

I therefore ask the authors of this report, which pretends to help us in allocating to sanatoria their proper place in the national scheme, to furnish at least some positive evidence that may help to neutralise the otherwise inevitable verdict against sanatoria as a means of helping the poor. Surely, the voluminous report compiled by the late Dr. Bulstrode upon sanatorium treatment justifies me in saying that there are no satisfactory statistics dealing with the

results of the treatment of the poorer classes in British sanatoria, which can help us in forming an opinion. We are compelled to look to Germany to guide us by her careful and laborious statistical analysis of sanatorium results. One may ask too, why it is that in France there has been no effort to establish sanatoria on any moderate scale for dealing with tuberculosis among the poor? Further, those familiar with Weicker's careful reports know that special care was taken to try the system under most favourable conditions. In Weicker's sanatoria the presence of tubercle bacilli in the sputum was almost a disqualification. At any rate, in the majority of cases received into Weicker's sanatoria tubercle bacilli were not found in the expectoration. Is it not, therefore, little less than gratuitous folly to take our instructions from men like Sir Clifford Allbutt, Sir Lauder Brunton, Sir William Osler, and Dr. Arthur Latham, who so flippantly minimise the importance of early diagnosis that they even presume to condemn my system of tuberculin dispensaries because I make early diagnosis by means of tuberculin an essential and indispensable feature of these institutions? When I urge the supreme importance of exploiting tuberculin in diagnosis at these special dispensaries, I am following the lead of such men as Koch, Cornet, Petruschsky, Spengler, Moeller, Pfuhl, Krause, Turban, Behring, Mitulescu, Roepke, Bandelier and hosts of others. Surely, those of us who have fearlessly investigated this important phase of practical medicine are safer guides than those who presume to judge us without such investigations? Until the medical profession seriously recognises the enormous value of tuberculin in the diagnosis of early tuberculosis, we shall be only too familiar with the catastrophes which must follow this foolish and flippant disregard of the most valuable weapon we possess in detecting pulmonary tuberculosis in the stage when treatment is likely to be successful. There are few sanatoria in Germany where this use of tuberculin is not fully recognised. There are unfortunately too few sanatoria in England where tuberculin is used as a diagnostic agent.

Theory of Intoxication.

These authors, misled by their views of "auto-intoxication," imagine that in the progressive stages of pulmonary tuberculosis there is progressive intoxication of the system by means of tuberculin. It is surely recognised nowadays that the severe symptoms in the later stages of pulmonary tuberculosis (fever, sweating, emaciation) are due not to tuberculosis, but to the secondary and mixed infections. These severe symptoms have nothing to do with tuberculin. This toxic theory, as simple as Simon, begs the whole question and presents a one-sided and wholly inadequate idea of the tuberculous process. The element of infection and the presence of living, virulent tubercle bacilli are wholly ignored, and mixed infection has no place at all. Accordingly, those familiar with the complex character of the tuberculous process can only designate the description of these authors as a misrepresentation of scientific facts. One or two illustrations will best explain my meaning, and will prove also the artificial and utterly misleading system of classification which has been adopted by these authors in describing the forms and stages of pulmonary tuberculosis.

Firstly, it is stated, "cases of consumption may be roughly divided into four classes: Class I., those patients who become infected and recover without knowing that they have been infected." In an earlier paragraph it is stated that, "practically every man or woman over thirty-five bears evidence of having suffered from tuberculosis." I ask whether any group of medical men are justified in describing cases in Class I. as cases of consumption? Such a loose, inaccurate and unscientific definition cannot be too strongly condemned by those who distinguish between healthy individuals and those who are consumptive. Next, I have seen not a few cases in which to-day "the dose of poison absorbed is not beyond the capacity of the defensive forces," and to-morrow, as the result of some trifling accident or complication, "Class I." suddenly passes into "Class IV." This is not the result of a progressive intoxication, but is due to an extension of infection. For example, I have seen

several cases of latent pulmonary tuberculosis producing no symptoms to attract attention, and yet within three weeks the patients, adults too, have died of tuberculous meningitis. In a smaller degree, and by less severe extensions, "Class I." may pass into "Class II.," and even into "Class III.," not by virtue of the progressive intoxication, but because the infection has spread discontinuously by the lymphatics or blood-vessels to near and distant parts. Thus too, slowly, and even without attracting attention, Stage III. of the disease may be reached without any symptoms of poisoning, such as the theory of these writers presupposes.

This loose, inaccurate, and misleading classification of the forms of consumption is nothing else but a gratuitous attempt to prop up the cause of sanatoria, which are tottering to their fall, while it may entirely mislead all except those who are intimately acquainted with the variable and uncertain nature of the tuberculous process.

They go further in their presumption, and, offering not a tittle of evidence, they seek to induce their readers, medical and lay, to believe that those of us who use tuberculin obtain our successes by treating only cases hypothetically designated as "Class I." and "Class II.," which would "recover without any treatment or by taking a holiday at the seaside." They themselves assume the result which it is necessary to prove. I challenge anyone who has studied pulmonary tuberculosis to tell us how long such particular cases will belong to "Class II." There may be "no insanitary or devitalising conditions; no lack of food," and yet the disease may rapidly hurry its victim into "Class III.," because the virulence of infection is a factor which cannot be estimated, eliminated, or disregarded. Thus, according to this classification, one and the same case might to-day belong to "Class II.," and "would recover if given a holiday at the seaside"; but to-morrow it would be "Class III.," and "need careful treatment in a sanatorium"; and next week would belong to "Class IV.," because "he is going to die whatever may be done for him." This is the sequence of events which too frequently follows upon the haphazard system of diagnosis and treatment which dispenses with tuberculin. The dominant

error in this classification is that the prognosis determines the diagnosis. I will furnish another illustration from the sad plight of a doctor, who unfortunately consulted physicians guided by the haphazard ideas of Sir Clifford Allbutt and Co. He consulted several doctors (sanatorium experts), who scouted the idea of tuberculosis, and declared that he was run down and should spend "a month at the seaside." He returned worse rather than better. Then it was recognised that the condition was more serious, and he was advised to have sanatorium treatment. While in the sanatorium tubercle bacilli were found. At the end of five months he was still worse, and left the sanatorium. Shortly afterwards he came to see me, when he was obviously in a late second stage of the disease. If he had come to me in the first instance, I should have promptly tested him with tuberculin instead of making a guess, which proved to be quite wrong. At that stage treatment with tuberculin would have restored him to health; as it is, the trip to the seaside and five months in a sanatorium have brought him to this perilous state.

These tragedies are much more common than are generally supposed, and while leading authorities like Sir William Osler speak slightly of the value of tuberculin in the early recognition of pulmonary tuberculosis and in treatment, the medical profession deserves to lose caste, and the unfortunate victims of tuberculosis are forced to play their tragic rôle "unhousel'd, disappointed, unanel'd." The wise physician desires rather to modify the prognosis by adopting treatment that will bring back the victims of the disease from "Class IV." to "Class III.," and even to "Class II." If he can even occasionally succeed in doing this, he will frequently succeed in bringing back cases in "Class III." to "Class II.," and even "Class I.," while he rarely, if ever, fails to prevent cases in "Class II." passing into "Class III." or "Class IV."

I have urged in my Weber-Parkes essay that the worse the prognosis the better the method that succeeds in saving the victim of the disease. Nothing has convinced me of the value of tuberculin treatment so much as the extra-

ordinary successes I have obtained in cases in the II.-III. and III. stages of the disease, even when complicated with tuberculous ulceration of the larynx. These results were published in *The British Medical Journal* of November 26, 1910. See page 384. I challenge any laryngologist to produce a similar series of successful results obtained by any other method. If one can restore to health by means of tuberculin victims in these advanced stages of the disease with the worst of all complications—tuberculous ulceration of the larynx—a *fortiori*, it becomes a relatively easy task to obtain successful results in Stages II. and I. of the disease. I have obtained these successes in cases that would be rejected by any sanatorium, and I have obtained successes in Stage II. of the disease which have not been approached in any records of sanatorium treatment. In Stage I. of the disease my results have been such that I unhesitatingly endorse the dictum of Professor Koch that in Stage I. tuberculin cures pulmonary tuberculosis with certainty.

It is for this reason that I must deplore the mere *ex parte* statements of men who have not themselves constantly, persistently, and courageously exploited tuberculin in treatment, and earnestly demand that a searching investigation of actual facts and authentic observations should be instituted by unbiased and competent authorities, so that the multitude of victims of this disease may not be thus cheated by mere assertion and prejudice out of the only system of treatment which offers hope and relief to the majority.

There are two other points that need discussion before I have done with this Report.

These authorities speak of the limitations of tuberculin. There is no remedy known which has not limitations. Injections of tuberculin cannot restore life to a dead man, and cannot always save a dying man. Tuberculin also has no effect except upon a tuberculous process. No one has been more persistent than I in pointing out that tuberculin may even do harm when other infectious organisms have found entrance to a tuberculous lesion. I insisted upon this point long before Sir William Osler paid any special attention to the rôle of mixed infections in the later stages of pulmonary

tuberculosis. Mixed infections generally, though not always, appear late in the course of pulmonary tuberculosis, and one of the best ways of dealing with these mixed infections is to anticipate them, and by means of tuberculin produce that state of fibrosis in the tuberculous lesion which not only checks the extension of the tuberculous disease in the lungs, but also forms the best barrier against the invasion of the organisms which cause mixed infections. Thus, the judicious use of tuberculin in Stages I and II. of pulmonary tuberculosis is the best way of obviating this particular limitation of tuberculin treatment. I am quite convinced that the routine use of tuberculin in the second stage of pulmonary tuberculosis is the best means of preventing mixed infections in all forms of open pulmonary tuberculosis. As for any other limitations, which these authorities are careful enough not to define or describe, they must tell me what they are before I can admit their existence or deal with them. Personally I know no limitations to the use of tuberculin in pulmonary and other forms of tuberculosis, provided the disease has not wrought so much destruction of tissue that recovery can only be brought about by a miracle. I have already stated, and I am quite earnest in my statement, that I know no contra-indication to the use of tuberculin except despair. Within the last year I have treated at the tuberculin dispensary more than one hundred cases of pulmonary tuberculosis in all stages, and apart from the cases of mixed infections, always in an advanced stage of the disease, I have been unable to find any limitation circumscribing the use and value of tuberculin. To be quite accurate, I am ready to admit that it is not wise, except with certain precautions, to use tuberculin in cases of well-marked renal disease. Renal disease is itself sooner or later fatal, and there is not much object in curing a tuberculous lesion when there already exists a disease which in itself is likely to be sooner or later fatal.

The other point I wish to discuss is the need for "constant, skilled medical supervision." Again these authorities do not define what they mean by "constant, skilled medical supervision," but I take it that, infatuated with the idea that

"auto-inoculation" by graduated exercises is the cardinal principle of treatment, they mean that the physician must constantly watch this empirical system of introducing into the circulation a sufficiency of poison from the diseased foci to stimulate the tissues to produce a sufficiency of antibodies to neutralise the effects of the disease. I am convinced that, by a system of graduated doses, which can be measured with mathematical accuracy by the subcutaneous injections of tuberculin, there is not the same need for what is termed "constant medical supervision." In our system experience has shown that it is enough to record the temperatures every three hours, and to weigh the patients at regular intervals, in order to work out the salvation of the victim. We teach even our youngest patients to take their own temperatures, and we instruct them to keep quiet if the temperature rises. By working on this system for many years in Australia and for more than a year in England, where I have treated nearly two hundred patients, I have found no difficulties which cannot be surmounted.

It is too often forgotten that in dealing with tuberculosis among the poor it is necessary to reduce expense to a minimum, and if there is but little risk in carrying out this system at a cost of, at most, £2 for every case treated, I see no reason for spending another £30 per case in order to secure this much-vaunted "constant, skilled medical supervision." If this money were given to us, we should spend it, not on this "skilled medical supervision," which does not greatly benefit the patient, but rather upon trying to introduce permanent improvements in the homes of the poor, not merely in bricks and mortar, but in supplying the ordinary necessities and comforts of life. It is only too clear to me that this "constant medical supervision" is much overrated, because for at least ten years it has been my custom to treat even well-to-do patients at a distance—even in the late stages of the disease—and I cannot remember an instance in which these patients were any the worse because I was not at their elbow every hour of the day to tell them to do this and not to do that. In fact, by this system of training one teaches the people themselves to understand the

importance of carefully watching the course of their temperatures, and the value of improving their health and nutrition by a proper and regular system of dietetics, which can be controlled in a practical manner by the weighing-machine.

Lastly, by a proper course of tuberculin treatment it is almost the rule that cough and expectoration cease, so that the victims themselves cease to be a danger to their friends and relations. Banielier and others, including myself, have proved beyond all doubt that tuberculin treatment is a much more powerful factor in converting infectious into non-infectious cases than the best sanatorium treatment. This transcendent advantage of tuberculin treatment, which is admitted by all the best authorities—Turban, Banielier, Koch, Cornet, Petruschky, Spengler, Moeller, Pfuhl, Krause, Behring, Mitulescu, Roepke, seems to have no weight with these scholastic philosophers, and yet it is perhaps the greatest advantage of all. Such a system benefits not merely the individual victim, but mankind.

The problem under discussion is a national one, and a wrong solution jeopardises, not thousands, but hundreds of thousands of lives. Public opinion will surely condemn an *ex cathedra* statement which is based, not upon the consideration of new facts and fresh evidence, but on preconceived notions. It will surely be asked why the authors of this Report have not taken the trouble to examine the fresh evidence which is open to everybody and absolutely accessible, since the tuberculin dispensary is in the heart of London. The authors are either biased and therefore not fit to be judges, or afraid lest the evidence may upset their preconceived theories. How else can one explain the outstanding fact that they have dared to belittle and sneer at the new system of tuberculin dispensaries without even a casual acquaintance with its real work and purpose. *Amicus Plato, Amicus Socrates, sed magis amica VERITAS.*

PREFACE TO SECOND EDITION

THE success of the first edition leads me to think that a second edition will not be unwelcome. I appeal especially to the general practitioners of medicine and surgery. In my judgment the more a good remedy is used with care, discrimination and wisdom, the better alike for physician and patient, and for the community. As an individual using tuberculin I can do little, unless I can persuade the medical profession that tuberculin is the best remedy for tuberculosis, and both can and should be used by every well-trained physician. To gain this end I gladly invite medical men to the Tuberculin Dispensary at 263 Kennington Road, London, where they may be able to learn enough in a month (two days a week) to use tuberculin for diagnosis and treatment with safety and success. By this means many, if not all, who suffer may find help and relief that is otherwise denied them.

W. CAMAC WILKINSON.

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PART I

THE GREATEST PROBLEM IN MEDICAL
SCIENCE

TUBERCULIN IN THE DIAGNOSIS AND TREATMENT OF TUBERCULOSIS

PART I

THE GREATEST PROBLEM IN MEDICAL SCIENCE

IF it is the function of the physician to cure disease, and thus restore the patient to health, a stupendous task lies before those of us who have to deal with the victims of tuberculosis. Tuberculosis is the commonest and the deadliest of all the diseases that attack the human race, especially in civilised countries, and pulmonary tuberculosis constitutes about $\frac{1}{11}$ ths of all forms of tuberculosis occurring in man. Undoubtedly the statistics of Great Britain and probably of other countries understate the prevalence of this disease. It is stated on the highest authority that in Berlin during those decades of life when man is at his best, that is to say, between the ages of 20 and 50, tuberculosis is responsible for one-third of all the deaths. In England alone there are more than 55,000 deaths recorded each year—more than 150 deaths every day. In New South Wales the records are not so dreadful, but still there are probably nearly 1500 deaths from tuberculosis each year, for statistical records understate the incidence of the disease. In tuberculosis the mortality statistics—the death-rate—alone give us no clue to the actual number of persons suffering from this disease. Mortality is not morbidity, and there is not any known

Prevalence
and Death-
rate of Pul-
monary
Tuberculosis.

proportion between mortality and morbidity in tuberculosis. It is for the physician to reduce the mortality; for the hygienist to reduce the morbidity. Tuberculosis does not always and inevitably cause death. Many suffering from tuberculosis, chiefly in the very early stages of localised disease, get well without the physician's assistance, and do not die of tuberculosis at all. There is no reason why these cases should not also get well under sanatorium treatment or specific treatment; and in such cases the recovery may be readily attributed to the particular method. *Post hoc, ergo propter hoc.* We must all plead guilty to this comforting method of reasoning. A still larger number may not get well, but survive for one, two, three, or many years before death brings the disease to an end. In these cases the morbidity may endure through years, and all this while the victim is more or less maimed or disabled. This represents an *economic loss*, that fails to be recorded in mortality statistics. Further, many cases of tuberculosis appear in the Registrar-General's report under more euphonious titles—pleurisy, bronchitis, enteritis, laryngitis, even meningitis, pneumonia, and typhoid fever—to which no social stigma attaches. Worst of all, pulmonary tuberculosis, far the commonest and most dangerous form of tuberculosis, exhibits its cruel malignity by laying its withering touch upon men and women in the very prime of life, and too often strikes down ruthlessly the fairest flower of the family. Thereby the economic loss is further intensified. The care, and often the savings, of devoted parents have been lavished upon their children, who have drooped one by one under the blighting influence of this insidious and deadly disease. However much we may dread cancer as a fatal disease, cancer has the saving grace of attacking its victims when the best part of life is over. Most of the victims of tuberculosis are in the beauty of youth or early maturity, and the very bonds of love and affection give this heartless enemy the best opportunity of showing its malignity. In women the most fatal years are from 15 to 35, in men from 20 to 40. These terrible features should make consumption the most-to-be-dreaded of all diseases, but, because its insidious onset deceives and disarms us, and its

slow progress gives us plenty of time to get accustomed to its presence, consumption does not impress the ordinary mind as the most potent cause of misery and suffering—of disease and death—in the human race. If one-tenth of the number of deaths due to tuberculosis were due to plague, or cholera, or small-pox, there would be a public outcry against the inadequacy and futility of our measures for protecting the public health against such epidemics. There are every year in England 50,000 deaths from tuberculosis. Imagine the state of public feeling if the *Daily Mail* could have such a heading as "a thousand deaths every week from plague in England." Yet this does not adequately represent the death-roll from tuberculosis at the present time; and that too, let us remember, although the source of infection is well known and has been proved to be limited in distribution. Is it, then, too much to say that far the greatest and most difficult problem awaiting solution at the behest of medical science in the name of suffering humanity is the prevention and successful treatment of consumption? It is the special function of the physician to reduce the mortality by improved methods of treatment; it is the function of the hygienist to determine how best to lessen the prevalence of the disease by a system of prophylaxis based upon a careful study of the specific causes and of the channels by which infection occurs. No doubt the functions of the physician and the hygienist frequently overlap, for the physician should be, and is, a powerful factor in the dissemination of knowledge that makes for prophylaxis; and the hygienist has vastly improved our methods of treatment by isolating the cause of tuberculosis and providing us with remedies and methods that have been evolved from a careful study of the nature and progress of the disease produced in animals by experiment. Without these experiments we should be still groping in the dark, and the treatment of disease would be still a hopeless, haphazard system of empiricism. Through the profound genius of the greatest hygienist the world has ever known, Professor Koch, specific remedies for the treatment of pulmonary tuberculosis have been discovered, but by the irony of Fate, Professor Koch has hitherto been

6 TUBERCULIN IN DIAGNOSIS AND TREATMENT

"cursed rather than blessed" for his transcendent labours in the cause of suffering humanity.

The term "consumption" may be used to connote pulmonary tuberculosis in its advanced and infectious form, and

Man is	this infectious form of pulmonary tuberculosis is
Essential	the essential, though not exclusive, source of
Source of	all forms of tuberculosis. Whatever opinions
Infection.	may be held concerning the relative frequency

of infection from cows in infancy and early childhood, pulmonary consumption has no such origin. The common disease, consumption, does not come to man from cows or bovines, but from man himself afflicted with the infectious form of pulmonary tuberculosis. Not long ago this was held to be a self-evident proposition. Just now this simple truth is somewhat obscured by the strange medley of opinions that crowd around the academic thesis of the relation of tubercle bacilli of the human type to those of the bovine type. The origin of pulmonary consumption is surely to be traced to the tubercle bacilli of the human type.

Kitasato has shown that in 152 consecutive cases in which he examined the character and strain of the tubercle bacilli in the sputum of human beings affected with pulmonary tuberculosis, no other than the human type of the tubercle bacillus was found. Kitasato obtained the tubercle bacillus in pure culture in every case, and by scientific methods, including the injection of cultures into rabbits, demonstrated that the bovine type was never found.

Stabsarzt Dr. Möllers examined 51 cases in the same way, and Dieterlen 50 cases with the same result. Dr. W. H. Park of New York obtained the same results in 273 cases, so that in the words of Dr. Möllers "the absence of bovine tubercle bacilli from the sputum of men suffering from consumption, which makes up by far the greatest bulk of all forms of tuberculosis, shows the correctness of Robert Koch's view that bovine tuberculosis plays but a subordinate rôle in the causation of tuberculosis in human beings."

There is also a tendency to exaggerate the importance and prevalence of bovine tuberculosis in children. Professor Gaffky in his careful report of 300 cases of children examined

post-mortem, showed that 10 per cent. had tubercle bacilli in the lymphatic glands; in 11 of them the tubercle bacilli occurred only in the mesenteric glands; in 17 of them only in the bronchial glands. In 55 cases out of the 300 the human type of tubercle bacillus was found. In no single case was the bovine type discovered.

Stabsarzt Dr. Rothe contributes a further series of 300 cases of children under 5. 24 per cent. had tubercle bacilli in the lymphatic glands. In 3 of them the bacilli occurred only in the mesenteric glands. In 5 of them only in the bronchial glands. In 29 of the cases the human type of bacillus was found. In but one case was the tubercle bacillus of the bovine type found. Thus we learn, firstly, that among Berlin children of the poorer classes treated in hospitals 20 per cent. were affected. The bronchial glands were more often affected than the mesenteric glands. But the striking fact established by these investigations is, that in 76 cases 75 have been infected through human beings and only one from bovines. In other words, among children under the age of six 98.68 per cent. had been infected through human beings and 1.32 per cent. through cattle. Surely these observations confirm to the full the view of Professor Koch that even in children the danger of infection from milk is quite subordinate to the danger of infection from human beings.

Thus scientific investigations in Germany, America, France, Norway, and elsewhere have shown that in no single well-attested instance (save Reitzke) has the tubercle bacillus of the bovine type ever been found in cases of ordinary consumption. In a singular manner the epidemiological evidence, which Kitasato has supplied to us from Japan, offers further convincing proof that consumption in man has its origin in man, spreads from man to man, and perpetuates itself through its microbe in the body of man. If this was not the proposition which Professor Koch threw like a bombshell among the members of the tuberculosis Congress in London in 1902, it was something extremely like it; and Koch's opinion, that in man the essential cause and source of tuberculosis is pulmonary tuberculosis in man, is far nearer

the truth than many will allow. A few words upon the prevalence of tuberculosis in Japan may be surely opportune, seeing that no other country can hope to furnish such striking evidence. In Japan, as Kitasato tells us, the native cattle do not suffer from tuberculosis, and neither children nor adults drink milk to any extent, and yet in Japan there is just as much intestinal and meningeal tuberculosis among children, and just as much consumption and other forms of tuberculosis in adults, as in the chief countries of Europe. Even intestinal tuberculosis and miliary tuberculosis are as common in Japan as in England. These plain facts prove that in Japan tuberculosis in human beings has nothing to do with bovine tuberculosis, and on this and other evidence we may rest assured that at any rate pulmonary tuberculosis in England has nothing to do with tuberculosis in bovines. As I wrote more than ten years ago, "If tuberculosis were eradicated from cattle, the prevalence of tuberculosis among adult human beings would not be perceptibly affected." In Japan the antecedent is absent, and the consequent is not affected. Logically, the consequent is independent of the antecedent. No doubt, in young children localised forms of tuberculosis, especially tuberculous disease of the mesenteric glands, and sometimes even of the cervical glands, and occasionally generalised and fatal tuberculosis, may arise from infection conveyed by the milk of tuberculous cows, but these forms of tuberculosis are absolutely non-infectious and have nothing to do with the spread of pulmonary tuberculosis in man. The epidemiological evidence from Japan immensely strengthens the force of Koch's contention at the London Congress. Upon the vexed question of the relation of tubercle bacilli of the human type to those of the bovine type and *vice versa*, one may at least say that it is impossible to overrate the difficulties that beset the way of the investigator; but he who reads and inwardly digests the infinitely careful and strikingly convincing array of experimental details marshalled by Kossel, Weber, and Heuss, as the experts of the German Commission (*Tuberculose-Arbeiten*, Heft 1, u. 3), must admit that two types of mammalian tubercle bacilli exist. Most experts recognise this fact, but

until this difference is even more widely accepted, most of us must remain mere partisans; and, after having carefully studied most of the contributions upon this stupendous question, I, for my part, unhesitatingly declare my allegiance to the practical view, emphasised by Koch, that the prevention of pulmonary tuberculosis demands that we should vigorously deal with the sources of infection existing, *not in animals, but in man* affected with the open form of pulmonary tuberculosis, who scatters the seeds of consumption broadcast in his neighbourhood day after day, week after week, year after year, unless he has learnt the hygienic method of coughing and the simple means of rendering his expectoration quite harmless to others. We must not confine our attention simply to the massive expectoration, which can be seen and avoided, but must remember also that even if the sufferer attends to the instructions with respect to this massive expectoration, there is also danger in the cough. The patient cannot help coughing, and with each cough he may be projecting into the air a regular fusillade of invisible germ-laden particles, which may float in the air and then reach the lungs of the healthy, and there sow the seeds of a fresh case of the disease. The prevention of consumption is a far greater question than its treatment, and is and should be the business of everybody. The treatment must be left to the physician and his patient. Prevention concerns everyone; and it may be wise in the public interest even to administer without reference to the physician or patient. It is true that almost one-third of all the deaths that occur between the ages of 20 and 35 is due to tuberculosis, so that all those who love and cherish their sons and daughters in the bloom of youth and maturity should take a personal interest in preventing the awful sacrifice of life that must otherwise be laid to the charge of this murderous and pitiless disease. Calculated on a mere £. s. d. basis—not a very satisfactory basis—the money loss to England must be represented by millions of pounds every year. Every adult, whether as head of a household, as a mother, as a wage-earner, is worth on an average £50 a year to the State. On this low standard 50,000 deaths represents a loss of several

millions. Nor can one forget not merely the energy wasted in the individual who suffers, but that also cast away by others, sometimes for years, upon one who is hopelessly doomed. But apart from this *f. i. d.* aspect, the ruthless destruction of fifty thousand lives every year by a disease which can be, and should be, prevented, and can often be arrested by special methods too long despised, constitutes a problem of the gravest national importance. The problem is a stupendous one; it is very difficult, very complex and many-sided; but I believe we are now within striking distance of the enemy.

Consumption, as we see it, is rarely a simple process of uncomplicated tuberculosis. It is a process, variable in its nature, in its clinical manifestations, in its morbid anatomy, and in its course; and these variations depend upon the virulence and dose of the tubercle bacilli, the state of the tissue, and also on the number and character of the organisms that complicate the simple process due to the action of the tubercle bacilli. These variations, in their manifold clinical phases, have been analysed by the more exact methods of scientific inquiry, and, in consequence, method and clearness replace the confusion and vagueness that characterised our hitherto conception of the disease. Such an analysis, even for clinical purposes, may exhaust the methods of bacteriological research. More than a quarter of a century ago Koch's monumental work defined once for all time the nature of tuberculosis with marvellous accuracy and completeness. More than this; ever since the searching genius of Koch has been devoted to this subject, our knowledge has increased by leaps and bounds. Let us remember that in his earliest experiments, Koch set his mind to discover not merely the specific cause of tuberculosis, but rather a scientific method of treatment, based upon a careful study of the nature and effects of the specific cause isolated in a test tube. Practically all the sound and useful knowledge we possess with regard to the nature, the diagnosis, the prognosis, and even the treatment of this disease has but one source. It is to Koch that we owe those principles upon which we base our

earnest advocacy of preventive measures. To him we owe the safe and sure means of diagnosis in all cases of doubtful tuberculosis. To him we owe the scientific methods by which we can at will analyse the complications of tuberculosis and also surely judge the course of the disease. To him we shall not look—and we have not looked—in vain for scientific methods of treatment. Not only is his transcendent work in the past some guarantee of the truth of his scientific observations, but, as a disciple of this man of genius, I am able to assert, after nearly fifteen years of careful and laborious observations, that the main facts of Koch's original communications upon tuberculin are absolutely sound. Until the truth about tuberculin is recognised, the problem of the successful treatment of pulmonary tuberculosis must remain in abeyance. As Koch then maintained, (1) *tuberculin is an invaluable and indispensable agent in the diagnosis of early tuberculosis, especially pulmonary tuberculosis; and, as I have seen in many scores of cases, (2) the early stage of pulmonary tuberculosis can be cured with certainty by means of tuberculin.* It is my object to bring forward evidence that, in my judgment, establishes beyond reasonable doubt the truth of these two profoundly important propositions.

PART II

THE PROBLEM IN THE LABORATORY

PART II

THE PROBLEM IN THE LABORATORY

CONSUMPTION has been studied by physicians since the days of Hippocrates and Galen, but, although Lamer and Virchow devoted their best energies to a description of the effects observed in the lungs and elsewhere, their elaborate descriptions threw no light upon the nature of this disease, and it is well known to most of us that Virchow

Experimental
Investigations
of Koch.
Nature of
Infection.

and his followers were not the first to recognise that Koch, by finding life in the tuberculous masses, solved the mystery that was beyond the ken of the eye or the microscope. Sceptical they remained long after Professor Koch had convinced the man in the street. Villemin had convinced himself and others, notably Cohnheim, by experiments on animals that tuberculosis was the result of infection, and therefore, in accordance with the germ theory of disease, was due to the action of a living micro-organism; but Koch, by his epoch-making labours, removed every vestige of doubt, and finally convinced even the sceptics by discovering the very cause of this widespread disease and exhibiting it "in splendid isolation" as a pure culture, consisting of millions of tiny organisms, upon the surface of solidified blood-serum. In his masterpiece, "*Die Etiologie der Tuberculose*," Professor Koch brought before our very eyes the tiny, slender, living germ called the tubercle bacillus, and convinced the scientific world, very much against its will, that this tiny germ "was the one and only cause of tuberculosis, and without it there could be no tuberculosis." Since then volumes have been written about this tiny organism, which was first recognised

through its property of being deeply stained by certain aniline dyes (methylene blue, gentian violet, fuchsin) and retaining this stain even after the addition of strong acids. The tubercle bacillus is acid-fast. Since that day other acid-fast bacilli have been discovered, some of them (Moeller's Timothee- and Gras-bacillus) being distant relatives of the tubercle bacillus.

More important still, experiments in the laboratory have shown that tuberculosis in different species of animals may be produced by several varieties of tubercle bacilli, each resembling one another in certain features, but differing from one another also in morphological, cultural, and pathogenetic characters. The analysis of these varieties has not yet reached finality, and it still needs the illuminating influence of more and more perfect knowledge leading unto truth before the chasm that divides the experts will diminish and disappear—to the credit of science and the benefit of mankind. It is generally acknowledged that there are two main kinds of tubercle bacilli—the mammalian, occurring in mammals, and the avian, occurring in birds; while the weight of evidence at present favours the view that there are two distinct types of the mammalian tubercle bacilli—the human and the bovine.

The human type occurs under natural conditions in man and some other animals. The bovine type occurs in bovines and in swine. Nevertheless, the bovine type may also occur in man, almost exclusively in children under the age of ten, who have been fed on unboiled cows' milk. *There is no evidence at all in favour of the view that the bovine type of bacillus has anything to do with pulmonary tuberculosis, a point upon which too great emphasis cannot be laid—and it needs a great stretch of the imagination to suppose that the bovine type readily assumes the human type in the human body.* In children the bovine type retains all the characters of this type; in adults even the bovine type may in rare instances occur in tissues, but does not cause a progressive form of tuberculosis. Naturally, if adults drink unboiled milk from a tuberculous cow, the bovine type of bacilli may mechanically reach the human tissues, especially

in the vicinity of the intestinal tract, but these bacilli seem to have no power of causing progressive tuberculosis. Adult human tissues enjoy natural immunity against this type of bacillus. There is no cause for wonder in this when we know that a certain breed of cattle, the Japanese, enjoys natural immunity against any and every kind of tubercle bacilli.

The question at issue is no mere academic question; it is a question of cardinal importance, because, if much tuberculosis in man has its source of infection in animals, prophylaxis demands the full recognition of this source. In children, certainly, there is an obvious risk of infection—though it is very difficult to measure the extent and intensity of the risk—and the prevention of disease from this source can be secured by either eradicating tuberculous disease from dairy cattle or by simply exposing the milk to a temperature sufficient to kill the bacilli (pasteurisation). If, however, as Behring persistently asserts, and would have us believe, the common form of tuberculosis in man—pulmonary tuberculosis—is also due to infection during infancy by means of unboiled cows' milk, not only does prophylaxis demand the precaution of treating all milk so as to kill the bacilli, but we are, according to Behring¹ in his truculent attack upon Flügge, relieved of the otherwise irksome necessity of compelling consumptives to be careful with their sputum and circumspect in their mode of coughing. Fortunately, very few authorities take His Excellency Prof. von Behring at his own valuation upon this phase of the question, for it seems to have settled down into the solid knowledge of the world that at any rate the commonest and most deadly form of tuberculosis—pulmonary tuberculosis—is due to infection conveyed from man suffering from the infectious form of this disease to his healthy relative, friend, or neighbour.

Infection requires certain conditions; it may be potential or actual. Actual infection requires that the infectious material should be carried from the sick to the healthy and should therein find suitable conditions for its growth

¹ V. Behring recommends "Formalin-salt."

and multiplication. So long as the infectious material is alive it is potential; but outside of living tissue it cannot multiply under natural conditions and may even lose its virulence and its life. Preventive measures aim at preventing potential from becoming actual infection.

From all these considerations the great outstanding fact is that the commonest form of tuberculosis in man, pulmonary tuberculosis, the source of infection *par excellence* for man, has nothing to do with the tuberculosis of cattle.

It is in the laboratory that we seek to learn whether in pulmonary tuberculosis the initial site of infection is in the

Site of Infection:	air-passages or elsewhere—in the small blood vessels of the lungs (Aufrecht, Orth); in the tonsils (faucial, lingual, pharyngeal), whence via
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the cervical lymphatics and lymphatic glands the tubercle bacilli reach the pleura and then lodge in the apex of the lung; in the mouth (Westenboeffer); or in the loose meshes of the lymphatic system of the bowels of the infant (v. Behring). V. Behring tells us in poetic language that "consumption is the last verse of the song which is first muttered in the cradle." Most of us, for very many reasons, cling to Koch's original view, which Cornet especially, and more recently Pfeiffer and Friedenberger, and Flügge have sought to establish by experiments on animals, that in pulmonary tuberculosis the initial lesion is either in the air-passages or in the lymphatics that drain these areas. After the striking and convincing inhalation experiments, recorded by Kossel (*Tubercule-Abstrakt*, Heft 1, n. 3), of the German Imperial Health Office, it will need a great deal of special pleading to dethrone or discredit this simple and reasonable explanation. Tubercle bacilli are tiny, solid particles, not always single, but often in clumps or masses, which may be readily carried hither and thither by themselves or in coarser particles of dust. As solid bodies they are subject to the same physical laws as other particles in the air, some of which, such as coal-dust, we can easily track to the lungs by the air-passages and through the lungs by the lymph spaces to distant parts and distant lymphatic glands. The deeply pigmented, coal-laden lymph spaces and lymphatic glands

are the invariable accompaniment of life in cities. Moreover, anyone who had the good fortune to see for himself the beautiful specimens exhibited by Birch-Hirschfeld at the Berlin Congress on tuberculosis knows that primary tuberculous lesions do occur on the surface of the air-passages, especially in those very branches that lead to that part of the lung—the posterior section of the apex—where tuberculous lesions so frequently begin. Further, we may track the course of other micro-organisms in the lungs, especially of streptococci, pneumococci, influenza bacilli, and staphylococci. These organisms enter the air-passages and may, like tubercle bacilli, be localised in the apices of the lung (Neufeld), though more often they infect the mucous membrane of the bronchial tubes widely and may thence be conveyed to the tissue-spaces and to the nearest lymphatic glands, and even finally enter the general circulation. All these migrations of tiny organisms and coal-dust may be literally followed with the eye by means of the microscope, and it seems a work of supererogation to look for any other explanation when this natural explanation actually stares us in the face. Ribbert's and Oehlecker's recent investigations clearly point the same way. Although in animals, especially rabbits and guinea-pigs, tubercle bacilli may stray by circuitous routes from the intestinal tract or the mesenteric glands even to the thoracic glands (Oehlecker), the enormous preponderance of bronchial tuberculosis even in children (Barthet and Killiet, Still, J. W. Carr, etc.) is most readily explained by infection *via* the air-passages (Koch, Coenet, Ribbert, Oehlecker, Pfeiffer), rather than *via* the alimentary canal (v. Behring, Orth). To children, especially very young children, crawling on the ground in the homes of the poor, where there may be a consumptive who knows nothing of the hygienic way of coughing or of disposing of his expectoration, the risk of infection through tubercle bacilli of the human type is *daily imminent*. One knows that these tubercle bacilli have been found in the finger-nails of these doomed infants.

Thus in the laboratory we may learn much of the nature of the infective agent and also of the usual channels of infection. Along its track, too, the bacillus often leaves

behind its own peculiar mark or lesion, sometimes here, sometimes there, in the air-passages (Birch-Hirschfeld), in the tissue spaces (Virchow), in the lung tissue (Virchow, Birch-Hirschfeld), or in the lymphatic glands. In any or all of these structures the slow decay (caseation) caused by the bacillus may go on, especially in the lymphatic glands (Petrushky), even in the lungs and bronchial tubes (Birch-Hirschfeld), *without arousing the suspicion of the individual, the parent, or even his physician.*

This is truly one of the worst features of this treacherous little enemy. It may lie in ambush for months and years, and in this *latent stage of the disease* the health, the strength, and the appearance may suffer but little. The disease may become thus quiescent at any time; it may relapse after months or years; it may reach a serious stage, involving

the greater part of a lobe, and end in perfect restoration of health. In spite of treatment of any and every kind, the disease may never relent, closely pursuing the victim till death. Without any treatment the disease may come to a standstill, and never again disturb the health. The most affectionate regard for logic may not restrain us from attributing success to our own small efforts and failures to the vagaries of the disease. Our memories seize fast hold of successes; we leave others to record our failures, or leave them unrecorded. Let us adopt what measures we may, more or less failure is the rule, and yet we are apt to place a high value on many of our methods. As a rule the individual seeks advice when the disease is in the second stage, often in a late second stage; sometimes the disease reaches almost the third stage under the very eye of the physician before its real nature is recognised. *Pulmonary tuberculosis is so dangerous and deadly because, in the majority of cases, it is well advanced before its presence is discovered.*

It is very common for a physician to fail to recognise the disease in the first stage, and it is not uncommon also for a physician to discover disease in the first stage when the lungs are quite healthy. (See cases diagnosed by medical men as early phthisis, which I proved by a severe test to be free from tuberculosis.)

Detection of
Disease.

This is a very important chapter. These facts must be stated and recognised before the inestimable value of tuberculin as a diagnostic agent can be fully appreciated. Further, nowadays, sanatoria are the fashion. All authorities upon sanatorium methods agree that it is only in the first stage that really satisfactory results can be obtained. Turban, whose authority no one disputes, tells us that of 419 cases, only 11 came for treatment within a month of the appearance of the first symptoms, 103 within six months, and the majority, 305, only after six months elapsed. In the first group (11) permanent results were obtained in 72.7 per cent., in the second group (103) in 67 per cent., and in the majority (305) only in 40.7 per cent.

After Koch's discovery in 1882, the test of tests was the demonstration of tubercle bacilli in the expectoration by certain stains. For many years this test dominated everything. Progress in knowledge has deprived even this test of most of its value. Valuable in certain cases in which the physical signs are absent or misleading, and in mild cases of mixed or concurrent infection, notably in influenza and pneumococcal and streptococcal infections, the discovery of tubercle bacilli in sputum by certain stains too often fails when it is most wanted. It fails as a test in early diagnosis, because the presence of tubercle bacilli in the sputum is itself a sign that disintegration of tissue has occurred. The great lesson of the last decade has been the paramount importance of detecting pulmonary tuberculosis in the first stage—before tubercle bacilli escape into the air-passages by the disintegration of the superficial tubercular lesion—while, in fact, the tuberculous formation is still closed, or, in other words, shut off from the external world, represented by the air-passages.

Moreover, Koch and his pupils have taught us that, in the great majority of cases, the tubercle bacilli play a relatively subordinate part in the severe forms characterised by fever, sweating, and wasting. By the light of Koch's methods, as applied by Kitasato and Pfeiffer, we have been guided to a clearer and truer understanding of the variable

Analysis of
Processes.
Mixed
Infection.

picture that pulmonary tuberculosis presents to us in its manifold stages and phases. I admit that among English authorities there is still a strong tendency to look askance at, if not to disregard and belittle, the modern view of mixed infection, secondary infection, and concurrent infection. The term "mixed" infection—so full of light and meaning—was first used by Koch's distinguished colleagues, Ehrlich and Brieger, in 1882, in describing a case of typhoid fever complicated with malignant edema, but Koch himself was the first to recognise the condition in pulmonary tuberculosis. In his own monumental work upon the *Etiology of Tuberculosis*, one reads thus: "The combination of bacilli and micrococci as they appear in this disease belongs to the mixed infections, the existence of which appears to be by no means rare." In this case the infection was rather a concurrent than a mixed infection. There was an invasion of cocci from an ulcer in the mouth, in a case of miliary tuberculosis, running a rapidly fatal course.

The rôle of infection independent of the tubercle bacillus—sometimes mixed, sometimes secondary, sometimes concurrent—occurring in any stage of tuberculosis of the lungs, may defy exact measurement by clinical methods; but nowadays it is hardly possible to overrate its importance when bacteriological methods are called to our assistance. Diagnosis, prognosis, and even treatment demand a careful differentiation of the elements constituting these other infections. A careful analysis of these elements by recent methods is an important, perhaps the most instructive, basis of classification. Physical signs may mislead us in diagnosis and prognosis; symptoms may help us to distinguish the acute, the sub-acute, and the chronic—the treatment varying accordingly—but a discrimination of cases by the resources of bacteriological science gives us a basis at once definite and trustworthy. The full meaning and importance of mixed infection did not develop until the genius of Koch, which had guided and inspired the investigations that culminated in the production of the diphtheria antitoxin, proposed a new method of treating tuberculosis on an equally scientific basis. The treatment of tuberculosis with tuberculin is specific. It

is of no avail except for tuberculous diseases, and, if other complicating conditions exist—if other micro-organisms are associated with tubercle bacilli in the morbid process—tuberculin may fail absolutely to assist or modify the morbid process; indeed, tuberculin may then do harm, and perhaps even hasten a fatal issue. I fear that many men have learnt to their cost and at their patients' expense, that tuberculin may be powerful for harm and powerless for good when the chief cause of the symptoms is not the tuberculous process at all, but the various infectious processes that may be quite beyond the physician's range of vision. Tuberculin treatment aims at a progressive process of active immunisation, radically different from the passive process, in which antitoxic serum is supplied ready made. The passive process of immunisation may be exploited with impunity and under any conditions; active immunisation, on the other hand, makes a larger demand upon the energy of the tissue cells, and requires that the cells and tissues should be in a relatively healthy state. In the presence of various micro-organisms, such as streptococci, the energy of the cells may be so depressed that active immunisation by means of tuberculin cannot be expected. Tuberculin treatment, therefore, has limits unknown in the antitoxic treatment of diphtheria. The failure of tuberculin in 1891 was due to a disregard of the limitations and restrictions laid down by Koch himself, and to a general ignorance of the rôle of mixed infection in pulmonary tuberculosis. It was not that Koch's announcement was premature; it was rather that medical men, by a bitter experience, discovered themselves exploiting a remedy without having had the training essential to success. Failure in their hands was a foregone conclusion, and tuberculin as a remedy for tuberculosis had a mere mockery of a trial. The key to successful treatment with tuberculin is a proper selection of cases by means of bacteriological methods, or, these failing, by a judicious use of tuberculin itself as a diagnostic agent. There is, indeed, no royal road to success in the treatment of pulmonary tuberculosis, either by specific, rational, or other methods. Each case requires skill, judgment, experience, and, in no small degree, patience; and

after all the best and most conscientious efforts may be baffled by the intervention of conditions that can be neither foreseen nor prevented. Indispensable to success is a thorough knowledge of the variable, complex, and treacherous condition, mixed infection. This mixed infection may even parade itself in the garb and trappings of health. As a rule, fever is the danger-signal, and strongly suggests a secondary or mixed infection. Yet, in spite of the existence of fever, there may be no mixed infection, as in acute tuberculosis and in those rare forms of acute gelatinous caseous pneumonia which Fraenkel and Troje have so well described. On the other hand, fever may be absent, and yet mixed infection, with all its evils and dangers, may be imminent. Nothing short of a careful and thorough examination of the sputum by the methods of Pfeiffer and Kitasato gives us unimpeachable evidence of the presence or absence of a mixed infection. Such an examination should and must go hand in hand with specific treatment. Other methods of treatment may be independent of this fundamental knowledge, but in tuberculin treatment an analysis of the infectious process at work is indispensable. It is impossible to judge fairly the merits of tuberculin treatment, if we disregard the element of mixed infection. Mixed infection made shipwreck of the tuberculin treatment in 1891. By a fatal fortuitous coincidence, tuberculin as a remedy for tuberculosis was tried when influenza raged in Europe as a violent epidemic. Altogether apart from tuberculin treatment, the high rate of mortality from pulmonary tuberculosis in the influenza years proves that influenza was a very dangerous mixed infection in tuberculous disease of the lungs. If, perchance, tuberculin had been used, the tuberculin was blamed. Tuberculin has been condemned mainly, if not entirely, on the investigations of fifteen years ago. It was not known then that, although tuberculin might fail, and even do harm when influenza or other infection complicated the tuberculous process, tuberculin might do nothing but good when such complications were absent. I was fortunate enough to use tuberculin in some cases before influenza invaded Australia, and my limited experience seemed to prove the undoubted value of tuberculin if

it were used under conditions prescribed by Koch himself. But, while tuberculin can and does act favourably upon the uncomplicated tuberculosis process, it may do nothing but harm in the presence of a mixed infection. The phases and stages of pulmonary tuberculosis largely depend upon the manifold combinations embraced in mixed infection. Let me say, too, that certain infections of the lungs, that have nothing to do with the tubercle bacilli, may simulate tuberculosis of the lungs. This is especially true of influenza, and of infections due to streptococci or pneumococci (Neufeld). Such cases cannot be benefited by tuberculin. In my own experience, two striking cases are worth recording: I made a post-mortem examination on a man who died of some obscure septic process. The sole obvious lesion was a small abscess of the size of a hazel nut in the apex of the lung. The pus yielded in the smear a streptococcus, and in the growth on agar a pure culture of streptococcus longissimus. From the spleen, which was swollen and engorged, I obtained a pure culture of the same organism. Is there room for doubt that the primary infection attacked the lung? Even more pertinent is the second case, of a brilliant young medical man who fell a victim to his excessive loyalty to duty. He consulted me once, and once only, for chest trouble with cough and expectoration. For some time he had had irregular fever—sometimes as high as 101° or 102°. Physical signs were practically negative; at most, there were some suspicious auscultatory signs at the right apex. The sputum contained no tubercle bacilli, but I obtained therefrom an absolutely pure culture of streptococcus longus. I was inclined to the view that he was suffering from a mixed infection of tubercle bacilli and streptococci. Cases enough are recorded in which the tubercle bacilli do not get into the expectoration in the early stages of these mixed infections. As the fever persisted, an injection of tuberculin could not be given. If it had been given, it might have been held responsible for the sad issue of the case. In spite of my advice, this man continued at his post for some weeks during as bad a bout of cold rainy weather as I have ever known in Sydney. Then he went to the country for some weeks. He

returned to town, and was carefully attended by his regular physician. I saw him but once, a month before he went to the country. He developed septic endocarditis, and died in the course of some months. Surely this was a case of primary infection of the lungs with streptococci, which spreads thence to the endocardium by the pulmonary veins. Is it not possible that in many cases of cryptogenetic septic endocarditis the infective agents enter the system by the air-passages? I know of no record of a similar history and nature. Thus, septic infection of the lungs may simulate pulmonary tuberculosis. Far more frequently these infections supervene upon the primary tuberculous process. Streptococci, diplococci, staphylococci, and micrococci are ever at hand to complicate the tuberculous process. Influenza bacilli and bacilli pyocyanei may also complicate. I have isolated pure cultures of the influenza bacilli from many cases of pulmonary tuberculosis. In the year 1905, on the other hand, I examined no less than nine cases of so-called influenza, and in eight cases obtained a pure cultivation of a streptococcus, in one case of *aediplococcus lanceolatus*. There had been, according to my investigations, no true influenza in this epidemic. One may call it influenza from its clinical characters; but the frequent complication of true croupous pneumonia is fair clinical proof that influenza bacilli had not complicated the process. Thus, acute, and especially chronic, bronchitis may be due to concurrent infection of the bronchial passages, causing apical catarrh, and this infection of the bronchial mucous membrane may end in a mixed infection. In mixed infection the streptococci are intimately and inseparably associated with tubercle bacilli in the tuberculous lesion, as Spengler and others have shown, both by special examination of the sputum and by examination of the diseased tissues, in fatal cases. The tuberculous lesion has, of course, disintegrated, and tubercle bacilli are also found in the sputum. A mixed infection may cause only mild symptoms, especially if there is much fibrosis of the lungs, or may suddenly cause the signs and symptoms of acute phthisis with bronchopneumonic exudation. This

is important, and teaches a further lesson. Streptococci and pneumococci may attack a tuberculous as well as a healthy lung; but in both, the fatal disease is the same. Is it right merely because tubercle bacilli are found in the sputum, to make the diagnosis of acute tuberculosis? This is often done, especially during streptococcal and pneumococcal epidemics. *Those who see only tubercle bacilli in the sputum may err egregiously in diagnosis.* Hemorrhage may complicate a pure tuberculosis or a mixed infection, and may thus be either the first sign of a simple uncomplicated tuberculosis of the lungs in which the hemorrhage may be even a favourable sign or the early symptom of a form of acute phthisis due to mixed infection, and running a rapidly fatal course. As a rule, the supervention of the mixed infection leads to wasting, loss of appetite, and sweating, accompanied by fever of varying degrees. The clinical picture changes when a mixed infection is superadded to the chronic tuberculous process. Then, the tuberculous lesion may extend rapidly and soften; tubercle bacilli may be numerous or scanty in the sputum; the expectoration is greatly increased, and swarms with other and various organisms. Kitasato's or Pfeiffer's method readily evokes order out of the chaos of this flora. In 1891 the effects of the mixed infection of various sorts—influenzal, streptococcal, diplococcal—were ascribed to tuberculin. The lesions to which Virchow specially directed attention in fatal cases that had been treated with tuberculin were actually the lesions that commonly occur in severe secondary infections when no tuberculin has been used. The simple, oft-recorded fact, that death occurred within a few days or weeks of the injections of tuberculin in 1891, proves incontestably that tuberculin was used in hopeless and totally unsuitable cases. Thus the brilliant success of the few was placed in total eclipse by the disasters that followed the rash and indiscriminate use of the remedy by the many. Ten years of patient work and careful observation have hardly succeeded in rehabilitating tuberculin among the useful remedies for tuberculosis. Professor V. Schroetter told the Congress in London in so many words, that the work upon tuberculin as a remedy in tuberculosis

would have to be done all over again. Those who use bacteriological methods to their full extent will be convinced in less than a year of the supreme importance of mixed infections. Those who disregard the lessons taught plainly by the best bacteriologists will hardly listen to any arguments of mine. "They have Moses and the prophets: let them hear them."

In the laboratory also we may store up a knowledge of the endless variety presented by the tuberculous lesions in every stage of the disease, and nothing but a fairly accurate knowledge of these varieties can aid us in forming even rough and often inaccurate ideas of the possible conditions that may be compatible with the physical signs we may be skilful enough to record on our charts. Without a knowledge of these lesions, some causing consolidation, some softening, and some excavation of the lung tissue, physical signs may mostly mislead us. The physician attempts with the aid of physical signs to picture for himself the gross changes produced in the tissues of the lungs and bronchial tubes by disease in its progressive stages. These physical signs, which we feel and see and hear, and not the conditions, which we can at best guess at, constitute the essential basis of all systems of classifying the stages of pulmonary tuberculosis; and unless we have some clear idea of the changes which tubercle bacilli and tuberculin, without and with the co-operation of various other organisms, may produce in the lungs, pleura, and bronchial tubes we may go hopelessly astray in our diagnosis by means of physical signs. Mere degrees in the change of shape, in the impairment of movement, in vocal fremitus and resonance, in the change in the percussion note, in the altered quality of the breath sounds, and in the varying characters of adventitious sounds, signify much or little according to the skill with which we can translate the language of physical signs into the objective conditions in the underlying tissues themselves. Physical signs are always hard to interpret, and leave plenty of scope for erroneous conjecture.

Here, too, we may specially urge that it is far more important *not to misread the signs of early disease than it is to*

make the most accurate guesses as to the size and site and contents of cavities. He who never misses an early stage, and never finds an early stage in a sound lung, should be the life-long friend of many grateful patients; he who gives the most surprisingly accurate forecast upon the nature and extent of a cavity will in most cases soon be able to test the absolute accuracy of his diagnosis *post mortem*, and neither he nor anyone else can help the patient much. In the former case, if the physician knows the right thing and has the sense and courage to do it, he saves his patient's life. In the latter, his satisfaction begins and ends at the *post-mortem* table. Accordingly, it is well to seize every opportunity of studying the earlier phases of tuberculous disease of the lungs, which are found so often in those who die of other than tuberculous disease. A study of the early stage of this disease in the laboratory can alone teach us the great lesson that early lesions may hardly cause any physical signs of their own, and may cause no special symptoms. Most important is it to bear in mind this *latent* or early stage, because in a very short time, and sometimes suddenly without any warning, a severe hæmorrhage may supervene, or a severe mixed infection or both these catastrophes may occur, and the patient may rapidly pass into a perilous state. I have seen several instances in young girls in whom the latent stage was overlooked or ignored by the physician, and the quiescent form has ruptured into the air-passages; and the tubercle bacilli, thus liberated from the localised lesion, have been scattered through the air-passages by aspiration and caused acute tuberculous broncho-pneumonia, with all the symptoms of galloping consumption. Such forms may at first be called pneumonia broncho-pneumonia, acute influenza, or acute bronchitis. Nothing but the early recognition of these latent forms and prompt and effective treatment by means of tuberculin can prevent these awful catastrophes. The symptoms may be trifling and may abate and pass away, deceiving everybody, even the physician; there may be respite for a month, a year, or much longer, but as a rule recurrence takes place sooner or later, and the last state of the patient is far worse than the first, for there is progressive destruction of

the lungs. The disease may progress in the lung, as I have witnessed many times, while the general condition of the patient—his weight, his appetite, and his strength—improve. Often this improvement is due to the abatement of the mixed infection. I have seen laryngeal complications arise, when both the patient and physician were well pleased because the patient gained 10 lbs.

But while Koch was studying the ways of this enemy of mankind in the laboratory, he was at the same time searching for weapons wherewith to fight and overcome this deadly enemy when once it had invaded the human body. These weapons, discovered by the genius of Koch, are laboratory products, called tuberculin. In my humble opinion, we hardly begin to study the clinical aspects and vagaries of pulmonary tuberculosis until we use tuberculin for diagnostic purposes, as a curative agent, and also as a means of checking our results after treatment. In treatment, tuberculin is eminently specific, and though its use may be compatible with mixed infections, it in no way affects these complications; but in the presence of mixed infection the cells and tissues may not be able to make use of the tuberculin in their own defence. My own experience may have been peculiarly lucky, for in no single instance in the space of sixteen years have I seen tuberculin do any real harm if used with skill and judgment in cases of mixed infections, and I have seen remarkably happy results from its use in apparently hopeless cases of pulmonary tuberculosis—even in cases of severe mixed infections.

Tuberculin is the general name for the toxic products of the tubercle bacillus grown upon the artificial media of the laboratory. It is not a serum. Its character and effects vary with the mode of preparation. The toxic products of the bacillus are the soluble, diffusible exo-toxins, and the endo-toxins of the solid cell-body. Old tuberculin is merely a glycerine extract of virulent bacilli—either as a simple filtrate (T.O.A., T. original altes.) or reduced by evaporation to $\frac{1}{10}$ its original bulk (T.A.). This latter preparation is the old tuberculin of Koch, invaluable and often indispensable in

respectively are so very marked that it is hard to believe that P.T. is merely P.T.O. evaporated by slow heat to $\frac{1}{10}$ its bulk. I should be very sorry to give any patient $\frac{1}{10}$ of a gramme of P.T. after a dose of 1 gramme of P.T.O. which had not had the slightest effect. After a dose of 1 gramme of P.T.O., $\frac{1}{10}$ of a gramme of P.T. may cause a well-marked reaction. The older preparations of tuberculin, especially P.T. and old tuberculin (T.A.) contain besides tuberculin various forms of albumen, albumoses, etc. There is no virtue in these products, and while they do no great harm, it is better, if possible, to get rid of them. When I say that they do no great harm, I mean, that in scores and scores of cases I have given these preparations in doses of 1 c.c. and more at a time without producing any disturbance at all. Still, Professor Koch, shortly before his death, was occupied in purifying the tuberculin preparations of these albuminous bodies by simply growing the tubercle bacilli upon Proskauer's medium. The product T.A.F. (Tuberculin Albumose frei) is the latest form of tuberculin prepared at the Hoechstfabrik, and I have been using it for some time. As yet, I am not in a position to say that one can obtain the same success with this new preparation as with the old. If this is possible, the tuberculin treatment will probably be much simpler. This preparation, tuberculin T.A.F., has all the other properties of tuberculin and can be used also for diagnostic purposes. It is quite a different preparation from Beraneck's tuberculin, and must be used like the old tuberculin in large doses. Other forms of tuberculin may be made from tubercle bacilli of the avian type, and even from the saprophytic forms of tubercle bacilli which Moeller discovered in certain grasses and in dung.

These laboratory products constitute the chief and most powerful weapons we possess in dealing with the stupendous problem of the cure and prevention of tuberculosis in all its forms. By means of tuberculin properly used we can detect pulmonary tuberculosis and other forms of tuberculosis in the very earliest stage; by means of tuberculin we can cure with certainty the early stage of pulmonary tuberculosis, as Koch told us seventeen years ago; and by means of tuberculin we

can measure the effect and permanence of a course of tuberculin treatment and learn whether the disease has been eradicated or is merely quiescent. In the latter case a further course of tuberculin, perhaps in larger doses or in a different form, may be needed. If the disease has been eradicated I am of opinion that the patient having been thus actively immunised is less liable to a second infection or re-infection than a normal person. It is hard to conceive what more is required of a remedy. In diagnosis, in treatment, and in prognosis tuberculin is the remedy *par excellence* in pulmonary tuberculosis.

The immense value of tuberculin as a diagnostic agent in animals has long since been admitted, and I can testify to its value in human beings (*see tuberculin in diagnosis*). In

Page 32, third line

This essential difference between T.O.A. and Old T., and between P.T.O. and P.T. respectively, lies in the fact that in the preparation of Old T. and P.T. filtration is not carried out until the glycerine bouillon, *still containing tubercle bacilli*, has been evaporated to $\frac{1}{2}$ its bulk at 70° C. Thus endotoxins as well as exotoxins are contained in these relatively stronger preparations.

Further details of technical preparation will be found in Meister Lucius and Brünig's admirable description of all the tuberculin preparations introduced by Professor Koch.

Tuberculin Treatment.

again inoculated by Koch. The second inoculation ran the

respectively are so very marked that it is hard to believe that P.T. is merely P.T.O. evaporated by slow heat to $\frac{1}{15}$ its bulk. I should be very sorry to give any patient $\frac{1}{5}$ of a gramme of P.T. after a dose of 1 gramme of P.T.O. which had not had the slightest effect. After a dose of 1 gramme of P.T.O., $\frac{1}{15}$ of a gramme of P.T. may cause a well-marked reaction. The older preparations of tuberculin, especially P.T. and old tuberculin (T.A.) contain besides tuberculin various forms of albumen, albumoses, etc. There is no virtue in these products, and while they do no great harm, it is better, if possible, to get rid of them. When I say that they do no great harm, I mean, that in scores and scores of cases I have given these preparations in doses of 1 c.c. and more at a time without producing any disturbance at all.

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The immense value of tuberculin as a diagnostic agent in animals has long since been admitted, and I can testify to its value in human beings (*see tuberculin in diagnosis*). In animals also the curative value of tuberculin in its various forms has been proved by Kitasato, Pfuhl, Spengler, and Beck. These experiments show that even the old tuberculin, which was hastily condemned not only as useless but harmful, has a decided effect in checking the progress of tuberculous formations, and in causing in them those very changes that are recognised as positive signs of the regression of the morbid process. In guinea-pigs, inoculated beneath the skin with virulent tubercle bacilli, an ulcer, that persists till death, forms in the second week at the seat of inoculation, and the animal dies in eight to eleven weeks. In such guinea-pigs treated with tuberculin soon after inoculation, the ulcer healed, and the animals lived five, six, seven, and nine months after infection, though the control animals died in six, seven, eight, and eleven weeks (Kitasato). Kitasato writes, "I was able to convince myself with the greatest certainty that even tuberculosis of the lungs had been influenced in a decidedly favourable way by tuberculin treatment." He adds, "In the case of those guinea-pigs, five in number, that are still alive seven months after infection, the inoculation ulcer is at length completely healed, the lymphatic glands, previously swollen, are no longer to be felt, the animals have steadily increased in weight, and they have in all respects the appearance of healthy animals." More interesting still, these animals were again inoculated by Koch. The second inoculation ran the

following course—Some days after inoculation the tissue round the seat of inoculation was swollen and indurated. In a week this indurated part was detached by spontaneous necrosis, and exposed a fresh-looking granulating surface. This ulcer was entirely healed within twelve days from the date of inoculation—an event that never occurs under ordinary conditions. The second infection had no further effect. These results were repeated by Spengler.

But the use of tuberculin as a remedy for tuberculosis has gone beyond the laboratory stage, and many observers (notably Rembold, Krause, Petruschky, Spengler, Turban, Camac Wilkinson, and later Moeller) not only speak well of the remedy, but do not record a single case of generalised tuberculosis during or after its use. I have long since come to the conclusion that so far from causing generalisation, tuberculin is the remedy *par excellence* to prevent generalisation. Rembold began as a sceptic, as did Moeller, but their own results soon converted them. Rembold set a good example in waiting for six years (I waited longer still) before he collected his evidence upon the value of the old tuberculin. Out of 70 cases 27 were cases of mixed infection. Of the remaining 43, 16 were in an early stage, 15 in the intermediate stage, and 12 in an advanced stage. Of the 12 advanced cases, two were improved at the end of six years; the rest died. Of the 15 in the intermediate stage, eight were alive at the end of six years; all of them improved; one case was cured. Of the 16 in the early stage, one died four years after treatment; the rest were alive, 3 greatly improved, 12 permanently healed—75 *per cent.* *cured*. In these cases judgment was withheld till six years had elapsed. What sanatorium, without tuberculin as a remedy, can show such a record? And yet I can show a still better record, because I have been able to use many forms of tuberculin, and I have not hesitated to use large doses. Krause, Petruschky, Turban, Spengler, Moeller, and others are able to furnish equally satisfactory results in pulmonary tuberculosis with tuberculin as a remedy.

Before we conclude this chapter on the "Problem in the Laboratory," we must needs describe the effects of

tuberculin as studied in animals in the laboratory and in man.

"Humanum est errare" is not an adage that pretends to make a virtue of mistakes, and least of all should the physician, who is true to himself, manifest a facile tendency to condone mistakes; but, nevertheless, the history of medicine, as we see it in text-books, which sadly need revision, even before they reach the affectionate hands of students, in large part consists, if not of errors, at least of a correction of errors. In medicine mental inertia, tolerant of error, has been a frequent stumbling-block in the way of progress, and in medicine, as in politics, too-often mental myopia restricts the vision to but one side—and that a small side—of a many-sided and complex problem. Where can we find, though we search the whole domain of medical literature, a better illustration of this inept mental complacency than in the common attitude of mind towards the many and difficult problems that have been created for us by the parasite which is the essential cause of tuberculosis? Years and centuries of labour, and piles of volumes devoted to the problem of the nature and origin of tuberculosis, helped us not at all until the genius of Robert Koch—just a quarter of a century ago—obtained his cultures of the tubercle bacillus, in splendid isolation, active and virulent, upon the surface of his newly-found medium of solid serum. Ever since that memorable gathering of the Physiological Society of Berlin on March 24, 1882, when Koch announced his brilliant discovery heralding a new era in the history of medicine, our knowledge has increased by leaps and bounds; and even now in all that concerns tuberculosis—its origin, its nature, its prevention, its treatment—we do well to look for light and guidance to the genius who has devoted the best years of his life to the study of this protean disease. Let us never forget that the discovery of the specific bacillus of tuberculosis was the first step towards those fruitful investigations of Behring, Kitasato and Wernicke, which culminated in the discovery of a specific remedy for diphtheria and tetanus. Having dis-

The Nature
and Signifi-
cance of the
Tuberculin
Reaction.

covered the specific cause of tuberculosis, Koch next set himself to search for an agent that would destroy the parasites in the living tissues. Years of incessant toil, during which he exhausted the vegetable and mineral kingdom in his quest, led him to the disappointing conclusion that all disinfectants that would kill the bacillus in tissues would kill the tissues also. Nevertheless, there are many amongst us who still claim that creosote or *Ac. pyro. pome* are remedies for pulmonary tuberculosis. Proceeding then on absolutely new lines, Koch made the experiments upon guinea-pigs with graduated doses of dead bacilli which have revolutionised our ideas upon infection and immunity. Therefrom he evolved the principle of specific treatment, discovering that the disease can be not only prevented, but cured, through the medium of its own cause. On November 13, 1890, Professor Koch gave an account of a substance which was able to "render animals immune against tubercle bacilli and to bring to a standstill the tuberculous process in animals." This new remedy for tuberculosis, afterwards named tuberculin, consisted of a glycerine extract of tubercle bacilli, evaporated to one-tenth of its bulk in a water bath, and then filtered through a Chamberland filter. The method used by Koch in preparing tuberculin is in vogue at the present time. The effects of tuberculin were chiefly studied in guinea-pigs. Strange to say, guinea-pigs, that are so susceptible to living tubercle bacilli, are extraordinarily tolerant of tuberculin. Even a dose of 2 c.cm. of tuberculin, injected subcutaneously, has very little effect upon a healthy guinea-pig; but if the guinea-pig be already tuberculous, in the fifth week of the disease or later, 2 c.cm. will kill the guinea-pig with certainty in 6-30 hours, and produces, invariably, in the tuberculous lesions, especially in the tuberculous foci in the liver, an absolutely typical picture. This, in itself, was a great discovery. Never before had any substance been known which acted at a distance with certainty upon diseased tissue, and diseased tissue only. Prof. Koch then gave himself a subcutaneous injection of .25 c.cm., and found to his cost that man is far more sensitive to tuberculin than guinea-pigs. Calculated according to body weight, man is 1500 times as

sensitive as guinea-pigs, and yet in man there is far greater resistance to infection with living tubercle bacilli. In man tuberculous lesions tend to be localised—they may remain localised and inert for months, years, even a lifetime. Koch thus describes the effect of a dose of .25 c.cm. of old tuberculin: "Three or four hours after the injection, pains in the limbs, lassitude, inclination to cough, difficulty of breathing, which rapidly increased; in five hours an unusually violent rigor, which lasted an hour; at the same time nausea and vomiting and a rise of temperature to 103.4; in 12 hours all the symptoms abated, the temperature began to fall, and reached the normal level next day. The pains in the limbs and lassitude persisted for some days, during which also the site of the injection remained painful and reddened." Many experiments showed that in man the limit of tolerance to tuberculin is reached with a dose of .01 c.cm. (ten milligrammes). Such a dose has very little effect upon healthy persons, or even upon sick persons, provided there be no tuberculosis. But, if the individual be tuberculous, .01 c.cm. will produce a severe general and local reaction. "The general reaction takes the form of an attack of fever, ushered in with a rigor. The temperature rises to 103° to 106°. The other symptoms are headache, pains in the limbs, great lassitude, loss of appetite, nausea, vomiting; sometimes even slight jaundice and a measly rash on the chest and neck. (Herpes is also common.) A severe reaction sets in early, in four or five, even in three hours after the injection, and lasts 12-15 hours or longer. In milder reactions the fever is delayed, occurring in 8-12 hours." The fever then may be of short duration, and, therefore, unless the temperature be taken every two hours, it may be overlooked.

The local reaction is best studied in lupus patches, in which the tuberculin picks out the diseased tissues and spares entirely the healthy and healed parts. Koch says: "Even before the rigor begins, the lupus tissue swells and becomes red. The swelling and redness last while there is fever and may reach such a degree that the lupus tissue becomes

"brownish red in places, and necroses. This inflamed area
 "is surrounded by a whitish zone nearly $\frac{1}{2}$ inch in width,
 "which in its turn is surrounded by a broad, deeply red
 "border. When the fever passes away, the swelling gradu-
 "ally subsides and disappears in 2-3 days. The lupus foci
 "are then covered with crusts of dried serum, which separate
 "in two or three weeks, leaving behind a smooth red surface.
 "The phenomena thus described have invariably developed
 "with a dose of .01 c.cm., if any tuberculous focus exists in
 "the body, and I therefore believe that tuberculin in future
 "will become an indispensable diagnostic agent. One will
 "thereby be in a position to diagnose doubtful cases of
 "incipient phthisis even when on account of the absence
 "of tubercle bacilli or elastic fibres in the sputum, or through
 "the absence of physical signs, it is impossible to come to
 "any conclusion concerning the nature of the trouble. Thus,
 "also, it will be easy to recognise tuberculosis of glands, of
 "bone, doubtful lesions of the skin, etc. In apparently
 "arrested cases of tuberculosis of the lungs and joints it
 "will be possible to decide whether the morbid process has
 "actually come to an end and whether or not isolated foci
 "exist, from which the disease might light up afresh, as a
 "fire from a spark that glows beneath the lifeless ashes."

Surely we may here marvel at the precision which guides his clinical instinct to exploit this rich fruit of his laboratory experiments. May I presume to add that even the reaction that occurs at the site of injection often gives a fairly safe clue to the existence of tuberculosis in the body? In healthy persons there is no change at the site of the injection; in tuberculous persons there is much swelling, redness, oedema and firm infiltration, sometimes very extensive; and often immediately around the puncture there is further a well-defined deeply red, raised and even slightly vesiculated area about an inch in diameter. This appearance develops best in the forearm, and therefore I make a practice of giving test doses in the forearm. Roux has lately suggested a percutaneous test with tuberculin, much after the manner which Spengler recommends in the specific treatment of those highly sensitive to tuberculin. Still later a 1 per cent.

watery solution of dried tuberculin applied to the conjunctiva has been recommended as a test for tuberculosis by Calmette. This reaction, general and local, to tuberculin, is specific, and occurs only when tuberculous lesions exist in the body. Certainly, reactions, even with fever, have been recorded in cases of actinomycosis, syphilis, leprosy, cancer, etc., but in none of these recorded cases has tuberculosis been *excluded with certainty*; and it is easier for one who has seen many hundreds of such reactions with tuberculin to imagine that tuberculosis was somewhere present than to explain how the reaction *can* be other than specific. Certainly, in large enough doses, as Professor Koch proved in his own body, tuberculin is a *poison*, capable, like other bacterial products, of causing fever and severe symptoms; but, besides these ordinary bacterial elements, tuberculin contains a substance which has a selective action upon tuberculous tissue, and thereby causes the specific reaction. In doses of .005 or .01 c.cm. tuberculin does not cause fever in a healthy person; but in far smaller doses (.0001 c.cm.) tuberculin acts upon tuberculous tissue in such a way as to release an active fever-producing substance. The action upon the tuberculous tissue is essentially inflammatory. An enormous literature has grown around this subject from which we learn that, under the influence of tuberculin, the tuberculous tissue becomes drenched with tissue juices and infiltrated with the amoeboid cells of the blood. This inflammatory oedema may lead to softening of the tuberculous tissue. Healthy tissue is rigidly respected. Now, while it is easy enough to describe accurately this extraordinary effect of tuberculin upon tuberculous tissue, as yet its intimate nature is a hidden mystery. Long ago von Babes maintained that the local reaction was the direct result of the addition of tuberculin to the tuberculin already existing in the tuberculous focus—a simple process of summation or cumulation. As yet we do not understand what is termed the cumulative action of digitalis; so that this explanation is not satisfactory. The reaction seems rather to be akin to the phenomenon of over-sensitiveness to specific poisons, which was first described by von Behring in his experiments with

diphtheria and tetanus toxins. He found that animals immunised by the isopathic method (by graduated doses of the specific toxins) exhibit in the tissues obnoxious to the toxins a marked over-sensitiveness, which he termed histogenic over-sensitiveness, although the hematogenic immunity, due to the presence of abundant antitoxin in the blood, protects the over-sensitive tissues against the toxin. This view is certainly strengthened by the paradox reaction which Kretz discovered in immunised animals further treated with an equilibrated mixture of toxin and antitoxin. A normal animal does not react at all to an equilibrated mixture of toxin and antitoxin—there is no production of antitoxin. On the other hand, an animal already treated with toxins and thereby immunised, responds to the same dose of equilibrated mixture with an abundant production of antitoxin. This discovery of Kretz has been utilised for the production of antitoxins of high potency. Further evidence in the same direction is yielded by Behring's observation that the total amount of toxin necessary to kill a guinea-pig diminishes if the toxin be given in divided doses. Hyper-sensitiveness is established the more readily by small doses. In like manner Locuststein and Rappoport, working under Moeller, claim that in a tuberculous man or animal the specific toxins cause the tissues to respond by this hyper-sensitiveness; and they argue also that this very over-sensitiveness to the specific poisons is an indispensable step towards artificial immunity. All who have used tuberculin have very early in their experience become very familiar with the fact that at the outset there is this over-sensitiveness to tuberculin; and even, if you like to call it so, a temporary fall of immunity. Wright has called this the negative phase, but in my opinion has misinterpreted it and been misled by it. He considers that this over-sensitiveness is the result of overdoses or too frequent doses. Nevertheless, if small doses be repeated the period of this over-sensitiveness may be prolonged indefinitely. On the other hand, if the doses be gradually increased the over-sensitiveness rapidly disappears. Moreover, if the degree of immunity may be expressed in terms of the highest dose of tuberculin that can be given without causing a reaction, one cannot expect any

high degree of immunity from the method of small doses at long intervals, which is the fashion of those who follow Wright. With my experience to guide me, I cannot conceive that small doses of tuberculin not increased to any degree can produce immunity in either early or late stages of pulmonary tuberculosis; indeed, both in early and more advanced forms of tuberculosis, when attempts have been made for various reasons, on account of heart disease, or pregnancy, or hæmorrhage, to carry out the system of small doses, or by gradual increases, the result has been just the reverse of what was expected. Either no immunity has been produced or a prolonged phase of over-sensitiveness appears, which postpones the development of immunity. The natural course of pulmonary tuberculosis, in which no doubt infinitesimal doses of toxin or other immunising factors enter the blood, without any advantage to the organism, supplies the experiment under natural conditions, which sooner or later ends in failure. The stage of hyper-sensitiveness is prolonged and the disease gets the upper hand. By increasing the doses and overcoming the over-sensitiveness, according to Koch's original plan, immunity is produced, as I have proved actually in scores of cases of pulmonary tuberculosis in every stage. I do not exaggerate when I say that I have seen this over-sensitiveness again and again, and I can hardly remember any cases, except advanced and hopeless cases, in which I was not able to beat down the over-sensitiveness, and, by proceeding to large doses, have not only caused all symptoms to disappear, but months and years afterwards I have tested these cases under most exacting conditions, and have obtained no reaction at all. What Wright and those who follow his plan can do remains to be proved. If his method also succeeds, I can but say that still another mystery has been discovered in the vagaries exhibited by tuberculous tissue towards small and large doses of tuberculin. Moreover, as I have never yet failed by the use of large doses in early cases of pulmonary tuberculosis to bring the patient to that condition, which would at least be described as a case of complete arrest of the disease, I am not disposed to adopt a method which, after all, has not been shown to be even of value in tuberculous disease of the lungs.

On the other hand, for diagnostic purposes there is reason in exploiting this phenomenon of over-sensitiveness. Even Koch's own method recognises this tendency to increased sensitiveness towards repeated doses of the same strength. He gives the following directions:—"In suitable cases, a dose of 2-4 mg. of old tuberculin is given. In weakly persons one begins with 1 mg.; in robust persons, if the lesions are not extensive, one may begin with 4 mg. If no reaction occurs after the first injection, one gives double the dose in two days. If a rise of temperature, even of only 1 deg. F. (½ deg. C.) occur, then the second dose should be repeated as soon as the temperature has become normal. It often happens that this second dose causes a much greater reaction than the previous dose of the same strength. This is a very characteristic effect of tuberculin, and may be looked upon as unmistakable evidence of the presence of tuberculosis. If 2 mg. fails to cause a reaction, the dose must be increased till the limit of 10 mg. is reached." Koch elsewhere says that two doses of 10 mg. given at proper intervals and causing no reaction proves the absence of tuberculosis. Other authorities are content with a maximum dose of 5 mg., others with 3 mg. Loewenstein brought forward at the Tuberculosis Congress in Paris a special method which aims at producing over-sensitiveness by repeated small doses of the same amount. In children he gives ¼ of a mg. every three days for four doses, so that the test occupies ten days or so—an objection in itself. In adults ¼ of a mg. is injected four times, if no reaction occurs, at intervals of three days. Loewenstein claims that a reaction is produced in the great majority of cases. If no reaction occurs he proceeds to 2 mg.—6 mg. and 10 mg. I can see no special advantage in this method. Three injections are nearly always needed, and by virtue of the over-sensitiveness caused by this repetition of the same dose, temperatures of 102 deg. and 103 deg. may occur. There is no virtue in this over-sensitiveness; and if the tuberculin test is merely the prelude to tuberculin treatment, this method of repeated small doses is distinctly objectionable because the over-sensitiveness delays the process of immunity. I, therefore, follow Koch's method. The symptoms of the reaction are in pro-

portion to the fever. A temperature of 99.5 may be sufficient if there are general symptoms of mild degree and a local reaction; and especially if there is pronounced edema and infiltration at the site of injection. If there be merely a slight rise of temperature—not reaching 100 deg. F., I repeat the dose as Koch recommends. If the temperature rises to 101 deg. F. the symptoms are more marked, headache, drowsiness, lassitude, loss of appetite, and often pains in the limbs. If the temperature be 102 deg. to 103 deg. F., the symptoms usually set in earlier, last longer, and are more severe. Severe headache, great lassitude, drowsiness, nausea, even vomiting. Herpes is also common in severe reactions, and then also there may be delirium, albuminuria and even hematuria. Even a temperature of 104 deg. F. may follow such a small dose as 1 mg. Always, too, symptoms related to the tuberculous part show themselves. In pulmonary tuberculosis, cough, increase of expectoration, in which for the first time tubercle bacilli may be found, difficulty of breathing, and pains in the chest. Râles, too, may be heard for the first time. Swelling and tenderness develop in superficial glands, and, no doubt, in those also beyond the range of vision, if they contain tuberculous deposits. It has long been known that tuberculin causes an increase of the eosinophiles and a temporary decrease of the neutrophiles.

An account of the tuberculin reaction, which is, after all, a reaction of immunity, would not be complete without a reference to the most recent work of Wassermann and Brück upon the nature of this reaction; although a somewhat severe criticism by Moegenroth and Rabinowitch clearly indicates some faulty links in the chain of evidence which is brought forward by these authors to prove that the tuberculin reaction depends upon the action and interaction of tuberculin as an antigen, and its antibody, styled by Wassermann and Brück antituberculin, existing in the tuberculous focus. This latest development in the biological and biochemical diagnosis of disease, by means of the products of immunity, has been applied with success to such widely different diseases as syphilis, gonorrhœa, leprosy, and small-pox. The biological

Wassermann's
Experiments
and Views.

principles which lie at the foundation of such functions of specific sera as precipitation and agglutination, of haemolysis and of certain processes of immunity, have been applied by Wassermann and Bruck to the phenomenon of the tuberculin reaction.

The essence of their method of sero-diagnosis consists in the proof of the presence of specific antibodies—by the absorption and disappearance of the complement. Specially prepared sera acquire specific functions by means of specific products, such as immune bodies (factors in the mechanism of immunity), precipitins, agglutinins, all of them of the nature of antibodies. These specific antibodies, mixed in definite proportions with the antigens, are able to appropriate the complement and thus render it inert.

The test of the disappearance of the complement is by means of the simple process of haemolysis. The phenomenon of haemolysis takes place in the presence of the complement when the blood of an animal—e.g. a sheep—is added to the serum of a rabbit, which has been injected from time to time with sheep's blood. If the complement be appropriated or in other ways rendered inert, haemolysis does not occur. Accordingly the failure of haemolysis indicates the appropriation or disappearance of the complement. This method of investigation depends upon a variation at will of the three main factors concerned in the well-known reaction of Pfeiffer. In this reaction the three necessary factors are the cholera vibrios, as antigens; the immune body as antibody or amoceptor, and the complement which normally exists in the organism. If any one factor fails, the specific bactericidal effect fails also. Accordingly, if in an experiment two of the factors are known to be present and the specific phenomenon (in this case dissolution of the vibrios) takes place, it is certain that the three factors were present. Thus, cholera vibrios may be at once distinguished from other vibrios by this specific reaction of immunity. It was later discovered that not only bacteria but bacterial products or extracts may also act as antigens. Making use of this development in sero-diagnosis, it has been demonstrated by mixing together in certain proportions and under certain physical conditions

of temperature, etc., the syphilitic tissues of man or infected monkey, a complement and suspected tissue, such as the lumbar fluid of a general paralytic or tabetic individual, that the complement has been absorbed, proving the presence of a substance in the lumbar fluid which has a specific affinity for the syphilitic antigen. This experiment yields further proof of syphilis as an etiological factor in the causation of general paralysis and tabes dorsalis. Wassermann and Bruck have exploited this method of biological diagnosis in order to solve the mystery of the tuberculin reaction. The tuberculin reaction is a reaction of immunity, and the scientific analysis of this reaction is of importance, since it should surely help us the better to use the various forms of tuberculin in the treatment of tuberculosis.

In the tuberculin reaction two obvious facts present themselves. In the first place there is some selective action exhibited by the tuberculous tissue, in consequence of which tuberculin is drawn from the blood and concentrated in the tuberculous focus. The other event is the inflammatory softening of the tuberculous tissues. The tuberculous tissue attracts the tuberculin atom by atom to itself. Even so small a dose as 1 mg. injected at a distance profoundly affects the lupus patch. In a man of average weight the tuberculin is diluted in the blood at least 5,000,000 times. Tuberculin so diluted injected into the lupus patch would have no effect. Accordingly the tuberculin does not produce this profound effect by merely bathing the tissues in this diluted state. Specific affinity between the tuberculous tissues and tuberculin can alone explain the striking fact. Such specific affinities are nowadays known to be constant phases of cell activities, and, if we translate these affinities into the language of Ehrlich's masterly hypothesis, we should say that the substance tuberculin is withdrawn from the blood and concentrated in the tuberculous tissue by means of a haptophore atom group—a specific antibody which Wassermann has called antituberculin.

This antituberculin in lupus tissue attracts and binds to itself the tuberculin circulating in the blood. We need not look far afield for the *raison d'être* of antituberculin in the

tuberculous tissue. Long since Ehrlich, in order to explain the tuberculin reaction, suggested the existence of three distinct zones in a tuberculous mass. The innermost zone lying nearest the bacilli is saturated with tuberculin and mostly dying or dead—the outermost zone is healthy and unaffected—while the intermediate zone is damaged by the bacterial products and over-sensitive. When tuberculin is present in the blood it reaches the tuberculous focus and, affecting neither the central nor marginal zone, concentrates its energy upon the middle zone, where it causes the reaction through the agency of the affected but still active cell elements. In this reaction probably the young cells of infiltration and the emigrated white blood cells play the chief rôle, and in this tissue the antibody antituberculin is formed by living cell elements in response to the irritation caused by the tuberculin secreted from the active tubercle bacilli. The antibody is ready to seize with avidity its antigen, the tuberculin, artificially introduced into the blood. Of course, a complement is necessary, but the complement is likely to be ready at hand in the presence of active leucocytes to complete this biochemical process. Thus far the process is relatively simple and the explanation is not far-fetched or unreasonable. Tuberculin must exist in and around the tuberculous focus, either through the living or dead bacilli *in situ*; and in accordance with the general principles of local immunity may well form its antibody, probably in the intermediate zone, in harmony with Ehrlich's explanation of the local reaction. Now, while it may be relatively easy thus to explain the first great fact disclosed in the tuberculin reaction, it is far from easy to explain thereby the severe changes that affect tuberculous tissue.

These preliminary considerations will pave the way to our appreciation of the delicate and difficult experiments which have been devised by Wassermann and Bruck to explain the nature of the tuberculin reaction. These experiments were designed to prove (1) the existence of tuberculin in tuberculous tissue, and (2) the presence of an antibody, antituberculin, as the reaction product of the antigen tuberculin; and Wassermann and Bruck claim to

have proved both points by means of biological experiments, based upon the fixation of the complement. In their original investigations Gengou and Bordet showed that if emulsions of bacteria and the homologous inactivated immune body (or amboceptor) were mixed together, under certain conditions, and the complement added, the complement was fixed or appropriated by the antigens and antibodies. The proof of the fixation of the complement was furnished by the failure of hæmolysis, even when red corpuscles and their specific hæmolytic amboceptors are added to the mixture. There is no free complement to complete the process of hæmolysis. The complement has disappeared. Wassermann and Bruck went a step further, and showed that if soluble extracts of bacteria were used instead of bacteria, the same fixation of complement took place. Similar fixation of the complement occurs in the phenomena of specific agglutination and precipitation. It was by a converse process that Neisser and Sachs used this method to demonstrate the presence, not of immune bodies or amboceptors, but rather of antigens. The great advantage of this modification is that in such diseases as syphilis, in which the specific organism has not been isolated, the presence of specific antibodies can be demonstrated in the serum of monkeys infected with syphilis and the presence of antigens in syphilitic organs. In this way also syphilitic antibodies have been discovered in the lumbar fluid of those suffering from general paralysis and tabes dorsalis.

In mixtures, supposed to contain antigens and antibodies, under proper conditions the disappearance of the complement proves that both these elements are present. Only in the presence of the two elements specifically related can the complement disappear. Thus, by varying the experiment at will, it is possible to prove the presence of antigens on the one hand or antibodies on the other. By this method Wassermann and Bruck have proved the presence of anti-tuberculin and tuberculin in tuberculous organs. The method consists in mixing together in definite quantities the antigen and antibody, and then adding the complement (guinea-pig's

serum). This mixture is placed in an incubator at 37 deg for an hour. In that time the complement is fixed. That the complement has been fixed is proved by adding to the mixture the elements necessary for hæmolytic, *viz.* the complement, that is to say (1) the inactivated serum of rabbit which has been previously treated with the blood of a sheep, and (2) the blood of the sheep (5 per cent.). The complement used is guinea-pig's serum, fresh and normal. The prepared serum of the rabbit is the hæmolytic amboceptor, and the sheep's blood is the antigen. After the complement (guinea-pig's serum) has been added to the original mixture of the antigen and antibody to be examined and the whole has been incubated for an hour, inactivated rabbit's serum and the sheep's blood are added.

If, then, hæmolytic fails, both antigen and antibody must have been present, because the complement is no longer able to cause hæmolytic. Nothing but antigens and antibodies specifically related to one another can appropriate the complement. Accordingly, if it is known that antibodies are present in the mixture, failure of hæmolytic proves the presence of antigens, and, in like manner, if antigens are known to be present, the failure of hæmolytic proves the presence of antibodies. If hæmolytic occurs, the homologous body is absent. We are now in a position to understand the method of proving the presence of tuberculin (antigen) and its antibody (antituberculin) in tuberculous tissues. In this way the tuberculous organs and tissues and the serum of man, cattle, and guinea-pigs have been examined by Wassermann and Bruck. Extensive control experiments must also be carried out.

Organs were removed with aseptic precautions and ground up in a mortar in salt solution containing 5 per cent. carbolic acid, then well shaken, and finally centrifugalised so as to get rid of solid particles. Next measured quantities of these extracts were added to diminished amounts of Old T. and T.R.E.m. Fresh normal guinea-pig's serum was added as complement, and the mixture placed in an incubator at 37 deg. for an hour. At the end of that time the hæmolytic amboceptor for the sheep's blood, and, lastly, the sheep's

blood itself was added. The mixture was again placed in incubator for an hour at 37 deg., and then kept in ice overnight. Table proves the presence of antituberculin in tuberculous organs.

PROOF OF ANTITUBERCULIN IN TUBERCULOUS ORGANS.

(a) Tuberculous Organs of Man.

1. Tuberculous Lung.

Excerpt of T.Lung	Old T.	T.R. Em.	Guerriglia's Arrest- Compos- mixture.	Hemolytic Arrest- serum.	Change's Blood.	Results.
Experiments	1	05	1	1002	5 %	Arrest of hemolysis (Antituberculin in Lung).
1	01		1	1002	"	Complete hemolysis.
1		05	1	1002	"	Arrest.
1		01	1	1002	"	Complete hemolysis.
Control	1	—	1	1002	5 %	Complete hemolysis—
—	05	—	1	1002	"	
—	—	01	1	1002	"	
—	—	—	1	1002	"	
—	—	—	1	—	"	
—	—	—	—	1002	"	
—	—	—	—	—	"	

Again—

- Lung extract + 1 T.—arrest.
- + 05 T.—arrest.
- + + 1 T.R.Em.—arrest.
- 1 T.—hemolysis.
- 1 T.R.Em.—hemolysis.

Explanatory.—T.R.Em. = Emulsion of dead tubercle bacilli.
Old T. = Old tuberculin. T. = Tuberculous.

Numerous experiments of a similar kind show that *antituberculin* is present in the tuberculous tissues both in man and animals. On the other hand, in thirteen cases of pulmonary tuberculosis not treated with tuberculin, no antituberculin was found in the blood—although in animals sometimes antituberculin was found in the serum. Similarly

it was proved that *tuberculin* was present in the tuberculous formations.

Wassermann next examined the blood of some persons treated with tuberculin and found antituberculin in the serum of persons who had tuberculous disease, though antituberculin failed to show its presence in the serum, when there were no tuberculous lesions. Hence we may conclude that injections of tuberculin in tuberculous persons cause specific antibodies to appear in the blood. Professor Ostertag found the same result after injecting tuberculin into tuberculous cows. Moreover, on mixing together tuberculin and antituberculin, no trace of precipitin appears, showing that the formation of antibodies is independent of precipitins.

We may deduce certain general conclusions from these observations. In the first place antibodies against the products of the tubercle bacilli are present in the tuberculous tissues, but not in the blood, and the tuberculin seeks out its antibody in the tuberculous tissues and thus produces the local reaction of tuberculin. The local reaction, ending in severe inflammation and softening of the tuberculous tissue, seems to depend upon this union of the antigen, antibody, and complement; but there are some difficulties in the way of attributing to the combination of antigens, antibodies, and complement the severe inflammatory changes of a local tuberculin reaction. Wassermann and Bruck take for granted that, whenever by means of amboceptors complements are concentrated and are active, albuminous substances in the organism are dissolved by a process of digestion. Hence they attribute the softening of the tubercular tissue to the concentration of complements brought about by the specific union of the products of the tubercle bacilli with their antibodies in the tuberculous areas, and there is some reason for supposing that not merely free enzymes in the blood, but substances formed from the leucocytes which crowd around the tuberculous centre, play an important rôle in this reaction. Arnet's great work upon the effect of doses of tuberculin upon the leucocytes of the blood in those suffering from pulmonary tuberculosis proves that many leucocytes are destroyed by doses of tuberculin, and in their death liberate

substances which play an important part in the production of immunity in pulmonary tuberculosis.

Again, in tuberculous persons who have been treated with tuberculin, antituberculin is formed in the blood, and, combining with the tuberculin given in treatment, protects the tissues from the action of tuberculin, and reactions fail. Nevertheless, the presence of antituberculin in the blood probably shows that the tuberculous process is not yet healed. In other cases treated with tuberculin, the doses of tuberculin do not cause antituberculin to appear in the blood. Such cases, which react to tuberculin as in health, may be considered to have been cured. It is very likely that these methods for determining the presence of antituberculin in the serum may influence one's methods of specific treatment. As yet we are only upon the threshold of inquiry. But in the light of their investigations, we may think with Wassermann that the specific reaction of tuberculous tissue shows itself because the products of the tubercle bacilli are drawn into the tissues by their antibodies, and by this process the vitality of the cells of the organism in digesting the tissue is concentrated at this particular place.

The value of this method has also been shown in cases of acute miliary tuberculosis. By the use of a tubercle bacillus immune serum and the serum of the patient, products of tubercle bacilli were discovered; and similarly, by using Old T. instead of the immune serum, antituberculin was found in the serum. This analysis of processes at work in the tuberculous foci further indicates the specific nature of the reaction. It teaches us clearly that a tuberculin reaction can only occur when the tuberculin penetrates the tuberculous foci, and there combines with its antibody. Accordingly, a tuberculin reaction cannot occur if there is no tuberculous lesion in the body, and fails to occur if an old tuberculous lesion has healed, or is so securely surrounded by fibrous tissue that no active process is possible. I have been thoroughly convinced for many years that one hardly begins to understand the nature of tuberculosis until one has studied it by means of tuberculin. In diagnosis we cannot do without tuberculin, in treatment we mostly fail unless we use tuberculin, and even in prognosis tuberculin

guides us aright in practice. I assert boldly that no one has a right to speak of arrest of pulmonary tuberculosis unless he has shown that the patient no longer reacts to tuberculin. Tuberculin controls our knowledge in a most exact way. Even with the best of treatment—of course I mean *specific* treatment—it is our bounden duty to control our results year by year by means of tuberculin. I am not satisfied with any treatment unless a year afterwards the patient is not only apparently well but fails to react to tuberculin. That is, in my judgment, the best guide to the degree of improvement and the value of treatment—*immunity against the toxic product of the specific cause*; and the larger the dose tolerated, the higher the degree of immunity. Using tuberculin in this way to check and control one's work, one may expect to succeed where others fail. Early diagnosis, direct and radical treatment by specific methods, and a trustworthy prognosis all depend upon the proper use of these various forms of tuberculin which have been freely offered to us in the cause of suffering humanity by the supreme authority in the world upon tuberculosis—Dr. Koch.

PART III

THE PROBLEM IN MAN, AS THE MOST
FREQUENT HOST OF THE PARASITE

PART III

THE PROBLEM IN MAN, AS THE MOST FREQUENT HOST OF THE PARASITE

It is obvious that the bacillus is not the disease; nor is even its presence in the human body equivalent to tuberculous disease. Tubercle bacilli may of course be found on any surfaces, exposed to the outside world, in the skin and beneath the nails, notably in children crawling about a dirty floor, in the nose (Strauss, 40 per cent.), throat, air-passages, alimentary tract, etc., without causing disease. Possibly in some men, as in some animals, *e.g.* Japanese cattle, there is natural immunity, or no disposition to the disease. If it be so, we have no means of discovering it or proving it. In most human beings there is a disposition to tuberculosis, if the tubercle bacillus but gets its own opportunity. This disposition depends upon an affinity between certain tissues or organs and the bacillus or its poison, or both, which leads to certain changes, called the reaction, in the tissues. This disposition may be general or local. In young children the disposition is general, and acute generalised tuberculosis is the result. In adults especially, in pulmonary tuberculosis, there is a local disposition, a tendency in the lungs, especially at the apex, to undergo these changes or this reaction in the presence of tubercle bacilli. The form of this reaction, its rapidity and progress, depend upon many factors, not merely on the tubercle bacilli, their number and virulence, and other organisms, but also on the site of the lesion and the state of the tissues invaded. No doubt, too, the age of the tissue (Behring), metabolic energy (Mitulescu), and even states of the circulation (Bier) and blood (anæmia, chlorosis) are subsidiary, and may be determining factors in the infective process.

If the affinity exists, there is irritation in response to the toxins of the tubercle bacilli, with the formation of new cells and positive chemiotaxis (leucocytic accumulation), followed by slow necrosis, due to endotoxins, called caseation. Inflammatory reaction is an essential part of the process, varying in degree, due sometimes to the action of the tubercle bacilli, but sometimes to mixed infection, or even to both infective processes. Caseation is the characteristic lesion of tuberculosis; and softening of this caseous area may have disastrous results, even when the lesion is small. It may lead to hæmorrhage; it may convert a closed into an open form of tuberculosis, with all its risks; it may lead to fresh foci of tuberculous disease at a distance from the primary focus; it specially favours mixed or secondary infection. I repeat that acute galloping consumption in young girls, acute tuberculous pneumonia, or broncho-pneumonia may thus arise, and may be called acute influenza, or acute bronchitis, until the careless or misguided physician rectifies his mistaken diagnosis. Worst of all, the caseous area may burst into a blood vessel, especially into a vein, and lead to acute miliary tuberculosis, resembling in its features the acute miliary tuberculosis produced in animals at will by an intravenous injection of small doses of tubercle bacilli.

These dreadful catastrophes will and must occur, until we learn to detect the disease in the earliest stage—a consummation devoutly to be wished, but quite beyond our reach unless in the interest of the unfortunate host we use tuberculin without stint and without hesitation for diagnostic purposes. It will at all times be hard to save the host's life in such cases, because not only the specialist in lung diseases must be ever on the alert, but also other specialists, and, above all, the general practitioner, who must be keenly alive to the danger of overlooking an early case of pulmonary tuberculosis. Pulmonary tuberculosis may masquerade in various guises and deceive the very elect. I have known several instances in which throat specialists have been content with the superficial diagnosis of laryngeal catarrh, or a hæmorrhage from the throat; gynaecologists have made the diagnosis of amenorrhœa and chlorosis; and other physicians have diagnosed gastritis,

gastric ulcer, rheumatism, hysteria, neurasthenia, when a few small doses of tuberculin would have revealed pulmonary tuberculosis in an early stage to be the efficient cause of *these symptoms of pulmonary disease in distant organs*. We learn often enough that throat symptoms, blood disturbances, digestive troubles, ovarian troubles, and nervous symptoms, *without any lung symptoms at all, may give the first clue* to the presence of pulmonary disease. It may be added that tuberculin alone can settle the diagnosis in early tuberculosis of the genito-urinary tract, of bones, of joints, and even of serous membranes, including the meninges. Once this great fact is realised and remembered at the psychological moment by every practitioner of medicine, and tuberculin is called into requisition to help the diagnosis, nothing but good can come to the infected host and honour to the physician, who is well worthy of his hire. In such cases which have come under my own notice, and there have been many, the rapid and extraordinary improvement of the patient under tuberculin would convince the most hardened sceptic. The bold use of tuberculin in these very suspicious cases settles the question whether there is or is not any tuberculosis, and one cannot express in words the enormous gain to the host of this insidious parasite if the disease should be discovered early and treated promptly with tuberculin.

Every individual has an interest in tuberculosis, especially pulmonary tuberculosis, because the disease is so common and so capricious in its incidence that infection may be the lot of anyone. A knowledge of the nature of infection may give us the power of warding off infection, and a knowledge of the nature and early symptoms of the disease may in some cases prevent that postponement of the visit to the physician which is one of the chief causes of disaster. After all, under our present system, it is the unfortunate victim who first discovers that there is something wrong, and he is wise to seek medical advice early. Too often the visit to the physician is postponed, and the disease is allowed to drift into the second or even the third stage. In my experience it is quite rare for the patient, in the early stage of the disease, to seek medical advice of his own free will. I have

a large array of early cases simply because I am convinced that infection occurs from person to person, and acting upon this conviction I proceed to test all cases that have been exposed to infection, especially in the family circle. The symptoms in the early stage are altogether too trifling to suggest serious disease of the lungs. Moreover, when disease in an early stage may or may not exist, even if the physician be consulted, two mistakes are possible. The physician may discover tuberculous disease (in his own opinion) when the lungs are free from disease, or he may fail to discover tuberculous trouble when it certainly exists. In my own experience I have discovered tuberculous disease of the lungs in scores of cases in which the disease has been overlooked by other physicians (or more often surgeons), and, on the other hand, I have proved it not to exist when the diagnosis of pulmonary tuberculosis (in various terms, "weak lungs," "tendency to consumption," "incipient phthisis," "a spot or patch in the lungs," "going into a decline") has been made. Such mistakes were inevitable and excusable in the olden days, when the diagnosis of early pulmonary tuberculosis was largely based upon vague guesses at truth. Nowadays, with tuberculin in our hands, whether we use it in the old or modified method of subcutaneous injection, by Pirquet's or Calmette's (Wolff-Eissner's) method, there is little or no excuse for these mistakes. Of course one has to admit that even with the most exact methods of clinical diagnosis human fallibility is responsible for some mistakes, and therefore at all times wrong diagnoses are possible, but such errors can be reduced to a minimum. Even with care, and after long experience, I plead guilty to one or two mistakes, but in every such case but one I should not have made the mistake if I had been allowed to conduct the test in my own way. In the one case (Miss L.) in which I gave a wrong opinion, I allowed myself to be influenced by the nervous irritability of the brother, and did not give the full dose as advised by Koch. In an experience of fifteen years, this is the only case of pulmonary tuberculosis in which I know that I made a mistake. Still, in order to establish confidence in this method of diagnosis introduced by Koch, I am ready to confess that if I had not

used this test as a matter of routine in my daily practice, I should have been guilty, like other medical men, of many scores of mistakes. The test, of course, demands that the patient should also carry out his part of the contract. The temperature must be recorded properly every two hours after the injection, and all conditions, such as going to concerts, etc., liable to disturb the temperature, should be eschewed. In order to diminish the risk of errors, it may be well to conduct the test in a sanatorium under a nurse's eye. Calmette's ophthalmic reaction makes the diagnosis perhaps easier, but in my experience the reaction in the eye may be severe, and therefore compels the patient to stay indoors during the test. Personally, I should, after my experience with Calmette's test, prefer a subcutaneous injection to an ophthalmic instillation. The old bogey of the danger of mobilisation after a proper dose of tuberculin seems to have vanished into unsubstantial nothingness whence it arose. From my experience I am bold enough to assert that far from tuberculin causing mobilisation, tuberculin must prevent it, because though I have seen many cases of generalised tuberculosis, chiefly tuberculous meningitis in the later stages of pulmonary tuberculosis, I have not had a single instance of tuberculous meningitis in the cases I have treated with tuberculin, although I have used enormous doses of tuberculin in every stage of pulmonary tuberculosis. I quite admit that in hysterical and neurasthenic subjects the subjective sensations may be extremely severe, and even the injection (*injection vacua*) may cause an attack of hysteria; still, by patience and firmness, it is not difficult to carry through a course of tuberculin up to large doses, in spite of hysterical outbreaks, and to beat back the disease. Once the real disease has been checked or cured, the functional disease also gradually withdraws. There is some justification for the view of those French physicians of a past generation who called pulmonary tuberculosis a nervous disease. The nervous symptoms may be largely the result of tuberculous disease of the lungs, obviously a toxic effect, although Koch, Wassermann and others hold that the toxins do not circulate in the blood to any extent.

Once pulmonary tuberculosis in the infectious stage appears in a family, the host must be made to obey strictly the regulations which are designed to prevent the infection spreading to other members of the family. The source of the infection is known, and provided proper measures are adopted by the patient, the risk of the disease spreading to the healthy is small; but otherwise there is a daily risk. It is in the homes of the poor that this risk is greatly intensified, especially in the later stages of the disease, when the victim becomes careless and often helpless, and then little children, crawling and playing about the floor, may easily become the innocent victims of a father's, or a brother's, or a mother's careless and even filthy habits. By coughing, or through the sputum, whether visible and massive or invisible and in minute droplets, such a patient may scatter broadcast the agents of infection, and often the injury he does to his nearest and dearest relatives is not traced to its proper cause. *The disease does not begin to show itself for one or more years, and after such a lapse of time the true source and cause of infection is quite forgotten.* It is the custom to disinfect the rooms occupied by such patients dying of the disease. Unfortunately, the damage is already done, and disinfection of this sort has but a partial effect. The door is bolted after the horse has gone. In the future it will come to be a *general practice to test those who for months and years have been exposed to the risks of infection.* Nothing has helped me in the early recognition of pulmonary tuberculosis so much as the routine use of tuberculin in these very cases. The disease is always in an early stage, and, in my opinion, can be completely cured by means of tuberculin in the great majority of cases.

The duty of the host towards his own family is obvious, as soon as the nature of the disease is established. Further, it is of the highest importance to determine whether the disease is so advanced that tubercle bacilli escape from the lungs during the act of coughing or sneezing, or even loud speaking, or in the massive expectoration. If the bacilli escape, special precautions must be adopted and enforced. I regret to say that even now, chiefly because heredity or a

constitutional taint is held to be the dominant factor in causing the disease, medical men do not instruct their patients how to act so as to avoid the awful risk of giving a brother, or a sister, or a friend the dreadful disease. *How can it be otherwise when physicians acquiesce in the conspiracy to keep their patients in ignorance of the nature of their trouble?* I can see no sensible reason for this *conspiracy of silence*. It is rarely fair to the sick, it is never fair to the healthy. Such silence can do no good, and may do great harm. I have always told patients the nature of their trouble, if there was any risk of infection, and in no single instance has the patient been depressed or injured by the simple statement of the truth. It is quite easy to gain the confidence of the patient by encouraging him to believe that he must get much better, and if tuberculin be used early and properly, this hope will be fulfilled. Besides, one must treat the patient as an intelligent being, not as a child, when one urgently needs his co-operation in any system of treatment or prophylaxis. The host of the parasite of pulmonary tuberculosis in an infectious form, commonly called consumption, has clear duties towards society that must be respected if he wishes to remain within that society, but it is necessary to restrict his freedom as little as possible consistent with safety to others. Beyond a doubt we have to face an awkward dilemma when we advise upon the question of marriage. The risk of marriage is greater for the woman than the man. The increase of duties and therewith anxieties in the household may depress vitality, but for the woman pregnancy is the chief source of danger. If not pregnancy itself, certainly the normal duties of maternity inevitably increase the tendency to recrudescence of a latent focus and quicken the activity of an already active tuberculosis. Certainly if the mother be suffering from pulmonary tuberculosis in an infectious form, safety for the child requires that a healthy wet nurse should be provided, for the child should not be exposed to risk of infection by its mother after its birth. How to secure safety to the child in such circumstances may tax the resources and judgment of the physician. The relief from lactation may save the mother's life, especially if at the same time tuberculin be used for curative purposes.

In the man, the chief danger lies in the possibility of infection spreading to the wife. There are those who think that this danger is not great, because unless the wife or husband, as the case may be, is predisposed, infection will not occur, and if either be so disposed, the risk from other sources is so great that tuberculosis in the husband or wife does not greatly add to the risk. My own impression is that the risk is distinctly and seriously increased. A serious difficulty in the way of a sound conclusion is the fact that infection may occur and not manifest itself for some years later. Further, one must not look for infection until the case has become one of open tuberculosis. Accordingly, all cases in the first stage are useless for the purpose of investigation. If tuberculin be freely used for diagnostic purposes, and von Pirquet's test may be used widely in order to throw light upon this phase of the question, we may have more definite facts upon which to come to a logical conclusion. Even if a wife or husband be expelling bacilli in the sputum, precautions may be adopted and diminish greatly the risk, or the dose may be so small that the evidence of infection is greatly delayed. Still, if one or other has suffered from open tuberculosis for two years or more, infection should be manifested in greater degree among these than among other married persons, who have not been thus exposed to infection in the family. Naturally, if a husband be consumptive, and others suffering from consumption be in the household, infection of the wife may take place, although the husband has not been the source of infection. In all such cases tuberculin as a diagnostic agent should be used, either in Koch's way or in von Pirquet's way, since infection may have taken place, although there be no evidence of disease in the form either of symptoms or physical signs.

Once a case of pulmonary tuberculosis—especially of an infectious character—has appeared in a family, the sufferer must be informed of the nature of his trouble, and very clear and specific instructions must be given to him not only as to the best way of disposing of his expectoration, but also with respect to his manner of coughing, etc. Coughing, sneezing, and even loud talking may be the chief source

of danger, especially when the cough is severe and frequent and the visible expectoration of small amount.

In such cases thousands of bacilli may be expelled into the air, and, yet being invisible and unseen, the imminent risk is not recognised. Whatever be the cause of the cough, *the handkerchief and not the hand should cover the mouth during the act of coughing*. Else the hand itself becomes infected, and in various ways may pass the infection on to others. How is it possible to put this reasonable system of prophylaxis into practice unless we take our patient into our confidence and tell him plainly the nature of his disease, with the double object of getting his assistance in every detail that pertains to his own good and the good of his fellow-creatures?

PART IV

THE PROBLEM FOR THE PHYSICIAN

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CHAPTER I

HEREDITY IN TUBERCULOSIS

THE discovery of the tubercle bacillus as the cause of tuberculosis has profoundly modified our ideas upon this phase of the problem. Heredity implies the transmission from parent to offspring of certain definite features, qualities, tendencies or potentialities, but no influence that comes into play after the union of the male and female elements, *i.e.* after conception, has anything to do with heredity. No one assumes that the male or female element may carry with it a tubercle bacillus. In fact, a disease due to a parasite cannot be inherited. The parasite belongs to the environment, which is absolutely outside of, and unrelated to, those vital qualities in cell-life which determine inheritance. A tuberculous mother may conceive, and the tubercle bacilli in her blood may pass through the placenta after it has formed and infect the fetus *in utero*, but this presupposes that the mother is suffering from a late stage of tuberculosis, when the tubercle bacilli are circulating in her blood. In such a case the fetus may be born with tubercle bacilli in its tissues, or even rarely with definite tuberculous lesions. This is not hereditary, but congenital tuberculosis. Such forms of tuberculosis have been observed in animals and man, but they are rare curiosities. It is physiologically inconceivable, if not impossible, that a tuberculous father can infect his offspring before birth in the act of conception. Long ago, Gärtner exhaustively discussed this phase of the question, and little further need

be said upon the subject. It is very rare for the tubercle bacillus or its disease to exist in the tissues of the foetus at birth, and in all cases the mother has been the subject of tuberculous disease during the period of pregnancy. In any case the hereditary transmission of an extraneous foreign substance can have no meaning, and it would be well if the term heredity in relation to tuberculosis were abandoned. At most one may imagine that tissues possess a greater or less affinity for certain germs or their poisons in accordance with Ehrlich's brilliant hypothesis. This tendency does depend upon inborn and transmitted qualities of cells and tissues, but similar tendencies no doubt exist in relation to many other, if not all, infectious organisms and merely imply vulnerability. No doubt certain qualities in the mother's blood, especially in the fluid part, may be transmitted to the foetus *in utero*, mostly increasing, rarely diminishing, resistance, but again these changes are acquired and not inherited. In the same way, the milk of the mother may convey to the suckling offspring elements that protect against infection, but then only if the mother herself has become protected by an attack of the special disease. These facts are merely the ordinary phenomena of natural and acquired immunity, and acquired immunity in itself implies disposition to disease. For the most part, acquired immunity is the direct result of a post-natal infection. Too often disposition to disease is a term which implies little else than exposure to infection. Von Behring goes further still, and is the author of the view that disposition to tuberculosis is nothing less than a condition depending upon the penetration of the intestinal mucous membrane of the infant by tubercle bacilli after its birth—is, in fact, tantamount to actual infection. Thus Behring holds the inheritance of tuberculosis to be merely a wrong name for infection in early infancy. Behring and others, notably Cornet, greatly discount the popular conception of predisposition. Many authorities still believe that a disposition to tuberculosis exists in certain races, and even in certain isolated families of the same race, quite apart from infection. Disposition is merely a relative term, indicating a bias towards infection just as natural immunity

indicates a bias against infection. In tuberculosis it would seem that, in spite of so-called predisposition, there may be a distinct bias towards acquired or artificial immunity. Thus many high authorities, Weicker, Turban and others, seem to prove that treatment in sanatoria yields better results in the predisposed. The predisposed resist the disease longer and resent it less than the normal individual. Tuberculosis, having its origin in an extraneous vegetable parasite, cannot be an hereditary disease, and, in default of the old doctrine, the disposition is variously interpreted. Some attribute it to an inherited taint, some to an acquired constitutional defect (Spengler, inherited syphilis), neither of which can be estimated or defined nor yet eliminated; while others again find the embodiment of this disposition in certain local anatomical features. Thus Birch-Hirschfeld traces the disposition to certain developmental defects in the apices of the lungs, W. H. Freund lays great stress upon certain anomalies in the thoracic wall, especially defects in the growth of the first rib, and Rothschild considers that abnormalities in the setting of the manubrium upon the sternum is a factor in disposition.

All these views (Behring, Birch-Hirschfeld, W. H. Freund, Rothschild) illustrate the progress of thought and increased knowledge from the vague and indeterminate to the definite and determinate. Thus dissatisfaction with the older explanations has led to a revolt against views that are as old, and should be as dead as Hippocrates. These iconoclasts, notably Behring and Cornet, will hear of no disposition to tuberculosis other than that which applies equally to diseases of a like kind, such as diphtheria, tetanus, glanders, leprosy or syphilis. The tendency of the old school is to labour the question of predisposition, the tendency of the modern school is rather to give a right value to the definite, tangible and measurable factor—the infective agent in all its variations and vicissitudes.

When we have mastered the specific affinities of the different kinds of tubercle bacilli in all their grades of virulence for different animal tissues, we may begin to understand the meaning of predisposition. Gradually our

present conceptions of disposition as a factor peculiar to tuberculosis will be brushed aside, like many another conception, in the clearer light of scientific investigation. Meanwhile there is nothing illogical in craving some consideration for the view that liability to the disease may be mainly the result of the opportunity for, and the intensity of, infection, and variations in the disease itself may depend rather upon the kind and virulence of the specific cause than upon the nature of tissue as a soil. The disease is the result of a specific affinity between the specific cause and the tissues, and this specific affinity manifests itself in the lesion. Whenever, then, a lesion occurs, there is evidence of specific affinity. The investigations of Schlenker, Naegeli and Burkhardt have shown that lesions held to be tuberculous are found in the majority of *post-mortem* examinations (Schlenker, 66 per cent.; Naegeli, 93 per cent.; Burkhardt, 91 per cent.). "Jedermann ist ein bisschen Tuberculös." Such latent lesions are found in the delicate (predisposed) and in the robust. But though these observers have recorded a great variety of lesions, many of them merely interesting as *post-mortem* curiosities, but having no interest for the clinical physician, they have ignored the tubercle bacillus. It is therefore not by any means certain how far these lesions have been misread and misinterpreted. Nor should we forget that these facts and deductions were drawn from observations upon the poorest section of society, living in a community (Zürich) where tuberculosis is very prevalent (Naegeli).

Further, it has to be remembered that more often than not, the rapid course of tuberculosis, which is relatively common in growing girls, is the result of other organisms (mixed infections) than tubercle bacilli; thus the clinical picture is further blurred by these extraneous factors. Are we ready, then, to extend our ideas of predisposition also to the infections caused by streptococci, pneumococci, influenza bacilli, etc.? These cases of galloping consumption arrest attention, and the acute course of the process readily suggests to the layman that there must be predisposition to account for this riotous behaviour. Since it has been proved that this tumult-

tuons and galloping form very often owes its origin to factors outside of the tissues and independent of the tubercle bacilli, it is evident that the logic of those who affect the hypothesis of predisposition must at times be of a sinuous or circuitous variety. The less we think and talk about predisposition, which we cannot define or explain, the sooner we shall find the path that will lead us to a practical solution of the momentous problem of the prevention and treatment of consumption.

Surely, too, it is rather absurd to speak of predisposition, on the one hand, as though it were a sort of predestination, and, on the other hand, affect to believe that residence in a sanatorium or in some lovely climate, even for a year, will effectually neutralise the action of this mysterious predisposition. Those who cling to this mysterious idea are the very people who applaud the virtues of climate, of high altitudes, of open-air methods, and add mystery to mystery when they attempt to explain the rationale of their "little systems, which have their day and cease to be."

It is quite conceivable that an injurious or toxic influence might be transmitted from the patient to the offspring, either through the male or female element, but such an influence might have no relation to the action of tubercle bacilli in the parents or ancestors. A specific toxic influence can only act when one or other parent is actually tuberculous—harbours in a living and active form the tubercle bacillus. But that which is designated predisposition to tuberculosis may have no relation to tuberculosis in ancestors. It would be hard for the theorists to explain how tuberculosis in a cousin or an aunt could thus affect collateral members of a family. Accordingly, I have systematically disregarded the history of tuberculosis in any but those in the direct line—parents, grandparents and their direct progenitors. If neither parents nor grandparents suffer from tuberculosis it is straining the conception of a constitutional tendency to breaking point to search for evidence among collaterals. Again, one may argue that this so-called constitutional bias towards infection may have nothing to do with the existence of tuberculous disease in parents or ancestors, and the occurrence of disease in the

"cousins and the aunts" is the visible expression of this bias. This is not the idea of predisposition that presents itself to most laymen and others who argue in a circle. The common idea is that a father or mother of tuberculous stock (whatever that may mean) can imprint upon their offspring a stamp or mark characteristic of tuberculosis, and as an illustration, the "habitus phthisicus" is the outward and visible sign of this tuberculous heritage. It has to be proved that this "habitus phthisicus" is in any way related to the existence of tuberculous disease in previous generations or parents. I have seen more than one case in which this "habitus phthisicus" was extreme in the child, yet both father and mother were quite healthy. Moreover, I have seen it well marked in individuals showing no obvious tuberculous disease. The external picture of the "habitus phthisicus" is well known. This "habitus phthisicus" manifests itself in the thin, weakly, woody, long, lanky individual with stooping gait and drooping shoulders, a narrow, flat chest, wide, oblique, intercostal spaces moving but little (paralytic chest), and wing-like shoulder-blades, projecting outwards. This type of individual is very thin, the skin is thin, and the vessels of the skin thin and weak. Often, too, the skin is cold and clammy, and cold drops of sweat often trickle down from the axilla; the hair of the skin is long, downy, light in colour, and poor in quality. Vasomotor irritability is marked, and flat-footedness is common. Such are the external signs of the "habitus phthisicus." This type, said to predispose to tuberculosis, may be independent of tuberculosis in parents or ancestors, may be absent in tuberculous subjects, and may certainly be, and perhaps is, the result of tuberculous infection in the individual. Naturally, such a form of chest, by interfering with movement (paralytic chest), eminently favours infection and the progress of pulmonary tuberculosis.

Because there is a liability to disease, it in no sense follows that the same disease in parents or ancestors has helped to create this liability. On the contrary, it is a general rule in infectious diseases in man and animals that the occurrence of infectious diseases in the immediately previous generations

tends to create some degree of immunity. This general rule may or may not apply to tuberculosis, but the simple fact, attested by the best authorities, that pulmonary tuberculosis runs a more favourable course and does not necessarily impair the health of the victim so seriously or so rapidly when there is a distinct family history of tuberculosis, seems to indicate a certain degree of inherited immunity. Thus the offspring of those families, in which tuberculosis has appeared in the previous generation, should be rather protected than predisposed. According to Turban, those with a family history gave better results than those without any such history in the proportion of 496 per cent. : 448 per cent. According to Weicker, in the proportion of 466 per cent. : 441 per cent. Thus the constitutional tendency does not add to the gravity of the prognosis in individual cases. The predisposed, in fact, stand the disease better, enjoy life better in spite of weakness and illness, and fight the disease better by living longer. In my own experience, the fine, healthy, active athlete, if he fall a prey to the disease, makes a very poor fight for life if he depends solely upon the vigour and vitality of his organisation.

From a sociological point of view, the question arises whether this disposition to tuberculosis may be related to a general weakness or loss of resisting power akin to degeneration. The physical deterioration of the European races has been a subject of anxious observation and inquiry of late years, but it can hardly be urged that the prevalence of tuberculosis is a measure of this physical degeneracy. In England, Norway, Sweden, Belgium, and even in Italy, the nation seems to suffer less loss from tuberculosis than in Germany, France, Austria, and Russia; yet the ugly facts disclosed by the recruiting officers of the English army, before and during the Boer war, offer us material rather for serious reflection than for congratulation. Tuberculosis is the special curse of cities, causing many more deaths in the city than in the country. Predisposition utterly fails to explain this. Opportunity for infection is the obvious explanation. At any rate, if the environment of cities be the potent cause, the disposition is acquired and not transmitted. Prisons,

asylums, factories—even convents and schools—favour infection, and the striking success that followed in the wake of preventive measures in such institutions in Germany shows that facilities for infection were the effective cause of the prevalence of the disease. The city population is recruited and reinforced from the country. Country life is the ideal life for the maintenance of a high standard of health in the nation, but if 60 per cent. of the male population of Great Britain shows signs of degeneracy, smallness of stature, narrowness of chest, and bad teeth, the country must also supply a certain contingent of degenerates. The colonies of Great Britain are trying to teach that health and contentment are the happy lot of the man who can live on the land. No doubt to the healthy instinct, country life with its abundant opportunities for physical exertion and life in the open air appeals with a force of its own, and perhaps when health and the faculty of enjoying these simple pleasures are on the wane, man lives but in a paradise lost, and restlessness and discontent may urge him to regain his paradise in the turmoil of the city. Thus the individual of weakly constitution may tend to gravitate towards the city, so that a certain number of degenerates found in cities may come from the country. Probably, too, by a process of natural selection, the weaklings and wastrels both of town and country seek those very occupations in factories, etc., that favour pulmonary tuberculosis. Thus far individual disposition may also discount the influence of occupation as a predisposing factor in the origin of tuberculosis. Now if tuberculosis mainly attacked weaklings and degenerates, some sociologists might question the wisdom of prophylaxis, but every medical man and very many laymen must know that, like other infections, such as pneumonia, small-pox, or typhoid fever, tuberculosis itself may and often does attack the best men and women in the race. There may be perhaps some grim satisfaction for the unfortunate victim of tuberculosis to bear this in mind. Nevertheless, not the most intense disposition can cause the disease unless the germ of consumption—the tubercle bacillus—has entered the tissues and found a lodgment. Even after their entrance, the germs may remain latent, or may cause

sufficient reaction in the tissues to lead to their imprisonment and render themselves harmless, at least for a time. These latent foci are a frequent source of trouble in the future. How long the tubercle bacilli may remain quiescent but still virulent cannot be determined. Lydia Kabinowitch records a case in which the nodule had become calcified and yet living, and virulent tubercle bacilli had survived therein. It is certain that the bacilli, though quiescent, may retain their life and virulence in joints, in bones, in glands, and in the lungs for many years, until some accident, a strain, a blow, a bruise, or an infection, especially measles,¹ or whooping cough, or influenza, or a simple cold, or a succession of colds, sets the spark to the infection, and active tuberculosis, even of an acute type, may suddenly assert itself. Then certainly the condition may be grave, and baffle the best efforts of the physician. Even tuberculin may entirely fail, because tuberculin must be given slowly and cautiously, and it may need many months to introduce a sufficiency of tuberculin to overtake the disease. Before these months have elapsed, even in the course of a few weeks, an acute process may run its course to a fatal issue. On the other hand, if tuberculin had been administered in the latent stage of the disease, such catastrophes might have been prevented. Even general diseases, such as diabetes and chloecosis, may give the sleeping enemy its opportunity. My own experience further convinces me that alcohol helps the disease to overpower the resistance of the tissues, no doubt by depressing vitality. If a tuberculous patient be addicted to alcohol, sometimes through the unwise recommendation of alcohol as a weapon for fighting the disease, I refuse to attempt treatment until total abstinence is ensured.

Hansemann relates a case in which tuberculous meningitis

¹ In a very striking way I have seen the effect of measles in increasing the multivirulence of tubercles, and this hypersensitiveness to tubercles is the manifest expression of increased disposition to tuberculosis. Case I. Woman in third or fourth month of pregnancy, suffering from extensive disease of right lung, and moderate disease in left. Treated with tuberculin: no reactions: had severe attack of measles. After measles, severe reactions, even with much reduced doses. Case II. Young girl, twenty, affected in just the same way. The attack of measles profoundly altered tissues in their behaviour towards tubercles.

supervened in a child soon after a serious fall on its head. At the *Post-mortem* examination, the *fons et origo* of the tuberculosis was found in a caseous bronchial gland at the hilus of the lung. It is a matter of everyday knowledge that an acute inflammation of the lungs, due to streptococci, pneumococci, the bacilli of influenza, and whooping cough, may convert a latent into an active tuberculosis of the lung. Until the true import of these latent foci of tuberculosis as frequent sources of active pulmonary tuberculosis be recognised, the full value of tuberculin in early diagnosis and treatment cannot be appreciated.

The enemy may lie in ambush for many years, but it is nevertheless a source of constant danger. A wise man will design to destroy the enemy before he has the chance of doing mischief.

CHAPTER II

SPECIFIC DIAGNOSIS

DIAGNOSIS is something more than giving a name to a disease. In pulmonary tuberculosis it implies a definite and certain knowledge of the cause of the disease (ætiological diagnosis) and of the effects of the disease as manifested, firstly, in structural changes (anatomical diagnosis), and secondly, in disturbed function (symptomatic diagnosis). The ætiological diagnosis is of the first importance, because structural changes are not open to direct observation, and the physical signs and symptoms, especially in the early phases of the disease, may be simulated in other diseases. The early diagnosis of pulmonary disease is quite the most important chapter in the problem before us. Like many other diseases, tuberculosis may be easily and completely arrested in its very early stage by proper methods. Nowadays, in infectious diseases at least, there is no room for "happy guessing." In diphtheria, in plague, and in other like diseases, diagnosis is something more than "happy guessing." There are means at our disposal in the diagnosis of infectious diseases which rarely play us false, but, before we consider these special methods of detecting the disease in its first beginnings, it will be well to have a clear idea of the early stage of pulmonary tuberculosis.

ANATOMICAL DIAGNOSES

But a few months before his death Birch-Hirschfeld demonstrated at the Berlin Congress Upon Tuberculosis that pulmonary tuberculosis most often begins as a local deposit in the mucous membrane of the medium-sized bronchus. In 32 cases of latent pulmonary tuberculosis occurring in 196

cases of sudden death examined by Birch-Hirschfeld, not one case presented the anatomical lesion of tuberculous broncho-pneumonia starting as a caseous inflammation of the finer bronchial tubes. In four cases the early changes consisted of an interstitial millary tuberculosis, while in 28 cases "the primary bronchial tuberculosis is by preference (*zuvörderst*) localised in the posterior half of the apex of the lung (more often in the right than in the left), and in the part of the upper lobe lying immediately over this—an area that corresponds to the distribution of the bronchus apicalis posterior. This part of the bronchial branch corresponds to the part of the chest wall which participates least in the respiratory excursions and presents also in adults, by virtue of its position, unfavourable conditions for overcoming mechanical hindrances to the respiratory air-currents. This disposition to disease is further favoured by certain defects in the development of these very bronchial passages that may probably be largely the result of unfavourable conditions of life during the period when the growth of the lungs is most active—in puberty." If primary tuberculosis of the lungs is due to the invasion of tubercle bacilli in the inhaled dust of the air, this anatomical disposition favours and determines the settling of the infective agents as foreign bodies. Conversely, these facts strongly favour the view that the infectious material is first conveyed to the lungs by the air-passages. Moreover, it is difficult to connect a disposition of the lungs, depending on anatomical defects or peculiarities, with any special constitutional tendency. Such defects may also be reinforced by secondary conditions in the lungs, catarrhal and other inflammatory conditions causing injury to the protecting ciliated epithelium. Once the tubercle bacilli have invaded the mucous membrane of the bronchus, further changes of a tuberculous character ensue, causing swelling of the mucous membrane, and narrowing of the lumen of the bronchus, followed in time, maybe, by obliteration of the bronchial tube. Thereupon the smaller bronchial tubes beyond the lesion may shrink and collapse, and produce the well-known signs of early pulmonary tuberculosis—depression above and below the clavicle, impaired movement in this

area, increased vocal fremitus, impaired resonance, altered breath sounds, and, maybe, the adventitious signs of catarrh. Herein we have presented to us by the hand of a master, not only the anatomical substratum, which produces the early physical signs of pulmonary tuberculosis in all their fine gradations, but also a rational explanation of the frequent incidence of tuberculous disease in this very situation. Such an anatomical lesion in its first beginning cannot fail to teach us why in the early stage of the disease physical signs may fail—one of the primary lessons to be learnt by those who desire to detect the disease in the very early stage. If we accept this series of anatomical pictures, based on careful and exact observation, as the condition obtaining in most cases of early pulmonary tuberculosis—a view in complete harmony with the less direct evidence of physical signs as observed by clinicians—we hold in our hands the key to the proper understanding of the meaning of the early stage of the disease and of the difficulties that may involve its detection at the bedside. Subsequent changes and extension of the disease to other parts of the same lung and to the lung of the opposite side are due to a variety of causes. I know of no better account of the usual course and progress of the lesion in the lungs than that given by Dr. Kingston Fowler in *Diseases of the Lungs*, by Kingston Fowler and Godlee, but I absolutely join issue with him upon the meaning and influence of mixed infections. It is quite evident that Dr. Kingston Fowler has not himself investigated this important phase of pulmonary tuberculosis. Once the primary focus has progressed to caseation, softening, and ulceration, the tuberculous process may extend on the distal side of the lesion, and tubercle bacilli, liberated from the original focus, may be drawn by the aspiratory action of the chest into neighbouring and different parts of the lung and even into the lung of the other side. Cough especially helps to scatter the germs in all directions, although at the same time it may get rid of much infectious material. The diseased area, especially if there be disintegration of tissue, however slight, indicated by the presence of tubercle bacilli in the expectoration, and obviously if there be much ulcera-

tion of tissue, offers a *milieu* pre-eminently suitable for the growth of other organisms, especially of streptococci, staphylococci, pneumococci, and influenza bacilli. Then begins the sad eventful clinical picture of the mixed or secondary infections. It may be at this stage when severe symptoms develop, that the victim is first conscious that he is really ill. Not seldom, even in this stage, the disease is misunderstood and misnamed "influenza," "a severe cold," "bronchitis," "pleurisy," sometimes even "pneumonia" and "typhoid fever," and further disaster may follow in the wake of slipshod methods of diagnosis. Any of these conditions may be present, but an aetiological diagnosis alone can bring the possibility of succour within reach of the patient and credit to the physician. At this critical stage in the patient's history there is no margin for mistakes. The urgent problem to be settled is whether there is infection with tubercle bacilli alone or in association with other organisms. This embraces the aetiological diagnosis, a subject quite important enough to deserve a chapter to itself, for herein lies the great secret of success. While the tuberculous focus is closed, tubercle bacilli do not escape into and by the spatum, and in this stage the risks of secondary or mixed infections are at a minimum.

Pleurisy is merely a phase and form of pulmonary tuberculosis—in most cases an integral part of the tuberculous lesion—rarely the initial or sole lesion. Such at least is the nature of the chronic pleurisy that occurs so frequently in tuberculous lesions at the apex of the lung. Elsewhere tuberculous disease of the pleura may occur and cause localised areas of dry pleurisy, or a chronic, acute, and sub-acute form of pleurisy with effusion. For years past I have tested with tuberculin every case of pleurisy that has come under my observation—cases of chronic latent pleurisy with abundant effusion, with moderate effusion, and other acute cases with high fever and severe symptoms, and so far every case tested (in fever cases after abatement of the fever) there has been a positive reaction. A few years ago Professor Osler admitted a percentage of 30 in these cases. I believe that now he fixes the percentage much higher. On the other hand I have seen many cases called pleurisy in which I could find no

physical signs and in which the tuberculin test was negative. One of these cases was treated at the out-patient department of a hospital for a year by two different medical men. The patient getting no relief, came to me. He had no cough, and there were no physical signs; he did not react to tuberculin; pain he certainly had, which was worse on moving, especially on twisting, but not on coughing, and affecting both sides. This was rheumatism, not pleurisy. Thickened pleura at the apex is a very common accompaniment of pulmonary tuberculosis and modifies the physical signs. These tissue changes at the apex constitute the chief basis of classification, especially in the cases in which there is no fever. If fever exists the cause of the fever must be determined if possible. In grave cases, fever—even high fever—may be due to the activity of the tubercle bacilli in the tissues, but in the vast majority of cases the fever occurring in pulmonary tuberculosis appears at a later period of the disease and signifies mixed, or secondary, or concurrent infection. Weicker speaks of a temperature within normal limits but showing marked subnormal depressions in the morning as suggestive of tuberculosis. The absence of fever does not exclude the presence of secondary organisms, for "passive mixed infections" are recognised. Further, since localised catarrhs, even at the apex, may be due to streptococci, pneumococci, and influenza bacilli (Neufeld), a confusion of non-tuberculous and tuberculous infections is inevitable, unless tuberculin is used for diagnostic purposes. In the presence of fever Calmette's ophthalmic reaction recommends itself. It is especially in cases accompanied by fever that the ætiological diagnosis is indispensable, and instead of waiting for the fever to abate, for the purpose of giving a test dose of tuberculin subcutaneously, one may proceed at once to use Calmette's method, or, better still, von Pirquet's method by vaccination. My own experience and the records of sanatoria show that within a year of the appearance of symptoms the disease may reach any stage. Hence the duration of the disease may deceive, for patients may allow the disease to reach the third stage before they consult a physician. Cases of this sort are mostly acute cases and are always accompanied

by fever and severe symptoms. Most often the acute symptoms, such as progressive weakness, emaciation, sweating, anorexia, and even diarrhea, are due to a mixed infection. The fever is generally intermittent, normal, or subnormal, 96-97° F. in the morning, 102°, 103°, even 104° in the evening (streptococcal curve). Less often it is more or less continuous, 102°, 103°, 104°, especially in the pneumonic form. I have not yet seen a case quite like those described by Fraenkel and Troje, and later by Spengler. Of acute cases I have had several, though even they are, fortunately, not very common. In these forms some localised lesion sets free a large number of bacilli which scatter themselves through the lungs and cause numerous foci which coalesce to form large areas of tuberculous infiltration and consolidation, followed by extensive softening, maybe a puriform softening of lung tissue. The tubercle bacilli are then specially virulent and cause the symptoms of an acute toxæmia.

SYMPTOMATIC DIAGNOSIS

There may be no pulmonary symptoms—no cough and no expectoration—even in relatively late stages of the disease. In one case there was extensive consolidation of the right lung, especially of the whole upper lobe, with no cough and no sputum. General symptoms—loss of strength and energy, and a state of invalidism due to the toxæmia—were pronounced. Cases may even reach a late second and third stage, although tubercle bacilli cannot be found in the expectoration. Although tubercle bacilli do often appear in the sputum early, it is wise to consider the stage relatively late, since there must be an area of softening with ulceration, and a closed tuberculosis has passed into the open form, with all its danger to the patient and risks to the relations and friends. On the other hand, the tuberculosis may long remain closed, chiefly during the early stages of the disease. Hence we must unlearn the teaching of some years ago (von Ziemssen, Strumpell, Grancher, Kingston Fowler) that the presence of tubercle bacilli in sputum is a very important aid in diagnosis. As a rule, when tubercle bacilli are present,

there is no difficulty in recognising the nature of the disease by other evidence; but too often tubercle bacilli fail to appear in the sputum when evidence of this kind is sadly wanted. Still, there is no excuse for failing to examine sputum, and if the result be negative, an expert's opinion may further ensure a correct diagnosis. The chief advantage of discovering tubercle bacilli in the sputum is that it *ipso facto* dispenses with the need of testing with tuberculin, and proves the necessity for precautions to prevent the infection of the healthy, especially in the family. In my judgment and in my experience failure to find tubercle bacilli in the sputum is a positive indication for tuberculin as a diagnostic agent. Those who neither examine the sputum nor use tuberculin are in a hopeless state with regard to the diagnosis of early pulmonary tuberculosis, and it follows therefore that when the sputum fails, or fails to contain tubercle bacilli, those who do not use tuberculin for diagnosis may either raise unnecessary alarm or encourage their patients to live in a fool's paradise. They are perched upon the horns of an awkward dilemma from which they cannot escape. If they trust to guessing, they might as well leave that to the patient; I have known so many wrong guesses, even by men who examine for admission to various sanatoria.

Certainly, symptoms may avail in diagnosis; they may be pulmonary or local and general. The general symptoms refer to (1) the blood and circulation, (2) digestive apparatus, (3) nervous system, or (4) in women to the sexual apparatus (amenorrhœa, etc.). Anæmia or, in young girls, chlorosis may be a part of pulmonary tuberculosis. Such girls are easily tired, or easily excited, and function of any sort is easily exhausted. There is a state of aglobulism with diminished energy, poor appetite, distaste for food, wasting, irritable weakness of vasomotor apparatus, irritability and increased frequency of pulse, especially on slight exertion, shortness of breath, palpitation, flushing, especially after meals, and marked sweating, especially in the axillæ. Persistent frequency of pulse (tachycardia) is a serious symptom, and sanatorium authorities reject those manifesting this ominous symptom; with a rapid pulse arterial tension is low. Indeed,

any or all the symptoms of a chronic form of anaemia or chlorosis are common. I have seen cases in which pernicious anaemia was suspected. Nevertheless there may be no such signs of anaemia or of circulatory disturbances, and increased frequency of the pulse is not often a symptom in the early stage of pulmonary tuberculosis. The temperature varies greatly, and must be taken every two hours for trustworthy records. The temperature may be normal in any stage, even in the third stage; it may be variable and unstable, easily disturbed by slight causes, e.g. menstruation, and is not usually high but sub-febrile (99.4° , 100° , 100.4°): A rise of temperature after meals and after exertion suggests early tuberculosis. At sanatoria the regulation of the exercise depends on the temperature. Fever after exertion indicates that the exercise has been too great. An inverse temperature is not common, but specially signifies tuberculosis. Severe fever may suggest typhoid fever, rheumatism, even malaria. Tuberculosis may cause symptoms simulating rheumatic fever and even swellings in joints (in one case erythema nodosum, as well as in another, erythematæ), like those of acute rheumatism. Those who have used tuberculin must be very familiar with the pains in joints and muscles that in certain individuals appear regularly after injections, especially in the earlier stages of treatment, often time after time in the same joint—in the hip, in the knee, in the thumb, in the little finger, in the heel—or in many joints, or in the back or bones, or in muscles (cramps in the calves of the leg, even agonising and paralysing); pains, too, in and about the thorax, quite apart from pleuritic pains, and at any rate without any signs of pleurisy. Less often pains in the neck and even in the eyes. There may also be severe pains in the epigastrium, doubling up the patient after each dose—perhaps related to diaphragmatic pleurisy, or they may be rather of the nature of visceral pains (ilead), or dragging, boring, and even acute lancinating or cutting pains in the shoulder, radiating into the arm, or deep pain in the chest. There are strange individual idiosyncrasies, because in the same patient the pains after injections are apt to affect the same part. I forbear to suggest that there may sometimes be a local

deposit to explain these pains. One has great opportunities for studying these pains—their varieties and situations—after tuberculin injections.

But more important than these general symptoms are the local symptoms in the lungs, especially cough, without or with expectoration. Cough is a common symptom apart from tuberculosis, but is one of the commonest and most significant symptoms of pulmonary tuberculosis in every stage. It is too often under-estimated by the patient and his friends, and should never be attributed to other causes by the doctor until he has absolutely excluded pulmonary tuberculosis by a searching examination. In the insidious form of pulmonary tuberculosis the cough may be the first symptom to attract attention. An early cough may give the physician the all-important clue to the real cause of the trouble. To call such a cough a stomach cough is folly; even among doctors the meaning and nature of this cough is often misunderstood. A slight cough, especially in the morning or after effort—maybe a mere dry, hacking cough—occurring in a growing girl or young man should not be lightly passed over, especially if the cough ends in retching. There are many causes exciting cough as a reflex phenomenon, but in early life, especially in young girls, when aneurism or tumour is not a likely cause, the wise physician should have no peace of mind until he has proved—not merely guessed—that the cough is not due to pulmonary tuberculosis. Quite apart from pulmonary tuberculosis, tuberculous glands in the neck or mediastinum may press on the endings of the vagus or on the trunk of the nerve and excite cough. Detecting tuberculosis in the glands, the physician may prevent pulmonary tuberculosis by promptly treating with tuberculin. I have had several cases of this nature. Signs and symptoms apart from cough may be entirely absent. To the specialist in diseases of the nose, throat, and ear, there are, of course, numerous familiar causes of cough, because the nerve endings of the vagus supply these organs, but he must beware lest he also misplaces the origin of the irritation. The specialist is somewhat disposed to trace every irritation to the area in which he "lives and moves and

has his being." I have seen so many cases in which first-class specialists, forgetting that sight cannot penetrate beyond the surface, have seen nothing to justify the diagnosis of tuberculosis, and yet, a year or two later, these patients have presented themselves to me in the second or even third stage of pulmonary tuberculosis. If nothing can be seen by the specialist, and the lungs have not been examined by an expert, this foolish guesswork may lead to a sad and fatal tragedy. The specialist in diseases of the throat must make these fatal blunders until he realises the enormous value of tuberculin in the early diagnosis of pulmonary tuberculosis manifesting laryngeal symptoms without any typical tuberculous lesions in the larynx. The hacking cough of chlorosis also cannot by its nature be distinguished from the cough of pulmonary tuberculosis, and, as chlorosis and pulmonary tuberculosis are often associated, nothing short of the ætiological diagnosis by means of tuberculin can settle the real nature and origin of the cough. The expectoration or phlegm which comes from the chest (while the "spit," a term in vogue in some sanatoria, comes from the mouth) varies in character and amount, and yields invaluable information of a direct kind. Quite apart from tubercle bacilli, or the organisms of mixed infections, the mere quantity and gross appearance may help in diagnosis and prognosis. During a course of treatment under tuberculin it is very interesting to observe how rapidly the amount diminishes and the qualities alter till there may be little or no phlegm, or it may be so scanty that one has to wait a month or two before one obtains enough for a single examination. In one case I waited two months for the expectoration, and although there was hardly more than enough for one cover-glass preparation there were a dozen tubercle bacilli in the field. In another very interesting case, which was being treated for pulmonary tuberculosis by a medical man who did not examine the sputum, I was surprised to find that the sputum contained a regular menagerie of bacteria, but no tubercle bacilli. I at once suspected trouble outside of the lungs, probably in the nose. There was no trouble in the nose itself, and no discharge; yet there was a tiny pellet of pus in the middle

fossa of one nostril, and on exploring the antrum with a cannula I washed out the most filthy putrid cloudy material. To make quite sure that there was no trouble in the lungs, I also tested with tuberculin, and the result to rot gr. Old T. was absolutely negative. Yet this woman had been treated for a whole year for lung trouble. The progressive changes in the sputum, with which we are all familiar, give most useful information concerning the state of the bronchial tubes. In the early stages the sputum may be watery mucus containing some charcoal pigment, and sometimes small opaque yellowish streaks or masses, which offer far the best field for the search of tubercle bacilli. Later, as the pyogenic organisms come upon the scene, the sputum becomes opaque and yellow or creamy, or of a dirty greenish colour. In rare cases this greenish or bluish colour may be due to the presence of the bacillus pyocyaneus. The number and character of the tubercle bacilli may not help much in the prognosis. Still, a large number of bacilli, distributed or in clumps, lying free in the mucus and not within leucocytes, is not favourable. On the whole, if leucocytes contain many of the tubercle bacilli, the prognosis may be better. The chain-like appearance of tubercle bacilli, in which the bacilli are generally wasted, notched, and irregularly stained, is most often found in a late stage when cavities exist. It is a fact, testified by many records, that even in the third stage tubercle bacilli may not be found in the sputum. In my experience it is exceptional not to find them. Of course, if there is bronchiectasis it may be hard to find the few tubercle bacilli in the abundant mass of expectoration. In such cases again a test dose of tuberculin may be necessary to establish the diagnosis. However, it is chiefly in the early stages of pulmonary tuberculosis, when prompt treatment by tuberculin can work such wonders, that the absence of tubercle bacilli from the sputum misleads the unwary physician, and he loses the golden opportunity of showing his wisdom and saving his patient from an imminent tragedy. It cannot be insisted upon with sufficient vehemence that in the great majority of early cases of pulmonary tuberculosis tubercle bacilli are not found in the sputum, and, therefore, an aetiological diagnosis

can only be achieved by means of tuberculin. Once this great truth, taught us so long ago by Professor Koch, is thoroughly recognised, the treatment of pulmonary tuberculosis becomes a relatively simple and satisfactory matter.

Hæmorrhage of the lungs manifests itself as blood in the sputum in every degree. Hæmorrhage may not occur at all in pulmonary tuberculosis, but the one predominant cause of blood in the sputum or hæmorrhage from the lung is pulmonary tuberculosis. Some specialists in diseases of the throat may think that bleeding from the throat is not uncommon. My experience tells me that bleeding from the throat can hardly be mistaken for hæmorrhage from the lung. If the blood comes from the throat, a specialist can easily settle its source by examining the nose from the front, or the naso-pharynx, by means of the rhinoscopic mirror, and fixing the site of the bleeding point. If he cannot find or see the bleeding point, and especially if the naso-pharynx is clean and dry, this source is absolutely excluded. In the pharyngeal wall or lingual tonsils and surrounding parts bleeding points may rarely be discovered. In a hospital experience of nearly fifteen years as a specialist in disease of the throat, I cannot call to mind a single case of bleeding from these parts which suggested hæmorrhage from the lungs, or, in other words, pulmonary tuberculosis. Always, too, after a hæmorrhage the sputum shows more or less marked traces of blood, and in time becomes rusty and brown. This does not occur in bleeding from the throat or simple blood-spitting from injury to the mouth or gums. Hæmorrhage, even small recurring hæmorrhages, but especially when the lost blood is an ounce or more, is always a serious symptom, and in itself signifies pulmonary tuberculosis until this disease has been excluded. I had two interesting cases of recurring hæmorrhage with moderate loss of blood amounting to 1 or 2 ounces a day. The physical signs at the apex were absent, but in both cases there were crepitations not at the apex, but over the lower lobe of the lung. In both cases the tuberculin reaction was absolutely negative. Hydatid disease was, therefore, diagnosed and the X-rays applied. In both cases a shadow was observed. In one case the lung was explored without locating any tumour.

but two months after the operation the patient coughed up the cyst and was soon on the road to recovery. In the other case I sent the patient back to the surgeon who had sent him to me, and, I believe, he advised the patient to wait till the X-ray picture was more definite. Hydatid disease of the lung is the only condition in Australia which must be remembered as a not infrequent cause of pulmonary hæmorrhage. Bronchiectasis and tropical abscess of the liver invading the thorax are less common causes. In the clinical forms of acute pneumonia with much blood in the sputum it is well to look for tubercle bacilli. Hæmorrhage as a symptom of pulmonary tuberculosis may be transient, and yet be the important clue to a correct diagnosis. It may be the sign of a mixed infection, when the occurrence of hæmorrhage is followed and may be preceded by fever in various degrees. This fever (103° , 102° , 101°) may persist for some time with remissions and intermissions. Such a hæmorrhage is a further sign that the lesion, perhaps hitherto closed, has become open. When hæmorrhage occurs early it often happens that then for the first time tubercle bacilli appear in the sputum, but tubercle bacilli may not be found in this bloody sputum. Of course the presence of elastic fibres in the sputum is not related to the early stage of pulmonary tuberculosis.

Laryngeal catarrh, with a tendency to colds in the head (naso-pharynx), may be an early symptom of pulmonary tuberculosis. Such catarrh is not tuberculous in character, and has, therefore, been termed pre-tuberculous. It causes hoarseness, which may be easily misinterpreted. I have found again and again in the history of my cases that in the early stages of their disease patients had consulted a specialist for the throat, and he had told them that they had catarrh, but it was not serious. The terrible sequel in the cases of at least a score of patients who consulted me a year or more afterwards should be an everlasting warning to those specialists who on a haphazard diagnosis called the early stage of pulmonary tuberculosis by the false name of laryngeal catarrh. Undoubtedly many conditions of the nose and naso-pharynx favour the development of tuberculosis, and the throat specialist should always bear this in mind. At

any rate he should avoid a wrong diagnosis, which may lead to a tragedy, by excluding tuberculosis in cases of laryngeal catarrh. If he fails to use tuberculin as a diagnostic agent in such cases he may be risking the life of his patient.

My own experience compels me also to direct special attention to the careless and often flippant way of dealing with pleurisy. In my own experience pleurisy rarely has relations to any other disease than tuberculosis, and its relations to tuberculosis are both frequent and intimate. Many and many of the tragedies due to pulmonary tuberculosis may begin with pleurisy, which will perhaps keep the patient in bed for a few days or weeks, but then grant him a long respite, even for many years. The recovery is so complete to all outward appearance and the respite may be so long that only those who know the vagaries of this disease will trace the subsequent tragedy of consumption to the trifling and transient attack of pleurisy. I admit that pleurisy is often diagnosed when it is not present, but more often these moderate, mild and insidious forms of pleurisy are discovered and treated as minor ailments, hardly worth the doctor's attention. I must protest against such a foolish conception of this serious disease. In the last few years I have taken the trouble to investigate by means of tuberculin every case of pleurisy that has passed under my notice either in hospital or private practice, and I seriously assert that so far I have not found a single case of pleurisy of this character that did not react to tuberculin. I have recently had a boy in my ward with well-marked signs of pleuritic friction. There was no temperature, yet he did not react to 100 cc. of tuberculin, nor was there any X-ray shadow indicating hydatids. Moreover, every case of pleurisy without a single exception was accompanied by other evidence of tuberculosis, and responded by way of improvement to tuberculin treatment in the most remarkable way. As soon as I meet with a case of mild or moderate pleurisy which is not tuberculous I shall consider it well worth recording. Of course, I am not speaking of pleurisy as a mere incident in other diseases, such as pneumonia, typhoid fever, influenza, or of purulent pleuritis, but of those mild or moderate forms

of pleurisy which so far have not received the attention they deserve and yet constitute an important contingent of the tuberculous diseases of the lungs. As soon as we begin to speak of pleurisy as a complication of pulmonary tuberculosis, we fail to understand the nature of pleurisy. A case of well-marked pulmonary tuberculosis without pleurisy would be well worth recording, and the invasion of the pleura, whether early or late in the progress of the disease, is the simple and inevitable result of the progress and extension of the disease. On the other hand, the pleura being near the surface, sensitive in nature, at any rate, if inflamed, and having special physical signs (friction), may just contribute the facts necessary for the diagnosis of tuberculosis, the prompt and proper recognition of which may save the patient from the later tragedy which too often terminates the clinical histories of "simple pleurisy." In my opinion when the signs and symptoms of these pleurisies are definite there is no need to test with tuberculin, because these simple and mild forms of pleurisy are always tuberculous. The acute form also, apart from the pleurisy that may accompany many infectious diseases, the so-called acute idiopathic pleurisy, is most often a manifestation of tuberculosis. Accordingly, if we use the term "pleurisy" as a name for a definite disease, we should mean nothing but a localised tuberculosis in the pleura. According to this view, our statisticians should further revise their tables, and medical men certifying to deaths from pleurisy should by inference know "pleurisy" only as a lesion of tuberculosis. If, then, the diagnosis of "pleurisy" is made, *ipso facto* there is tuberculosis, and treatment should be just the same as the treatment for pulmonary tuberculosis. Indeed, in most cases there is pulmonary tuberculosis with the pleurisy. Is it not strangely inconsistent to treat pleurisy as a slight ailment and to make such a to-do with open-air treatment and the like for pulmonary tuberculosis when they are virtually one and the same disease? This is my view of the matter, and it is based upon my own observation that what is commonly called pleurisy is virtually tuberculosis of the lungs.

A long chapter might be devoted to the aberrant symptoms of pulmonary tuberculosis, symptoms related not to the lungs

at all, but to other organs. Chlorosis, anemia, and amenorrhœa may be the striking symptoms which bring the patient to the doctor, and yet the physician or gynecologist may rest satisfied with an examination of the blood or reproductive organs, while the source of the trouble lies undiscovered in the lungs. Again, any form of dyspepsia, gastric insufficiency, gastric irritation or nervous dyspepsia, chronic ulcer of the stomach, gastritis, may be simulated by pulmonary tuberculosis. The immediate and permanent improvement obtained by means of tuberculin in many cases which have come under my observation constitutes a very satisfactory chapter in diagnosis and treatment. In all tuberculous forms of dyspepsia and gastric troubles, especially in young girls, tuberculin does wonders, both in diagnosis and treatment. Nervous symptoms may also be obtrusive in pulmonary tuberculosis. Such symptoms may be due to metastasis causing actual tuberculous lesions in the nervous system, especially in the meninges.

ÆTIOLOGICAL DIAGNOSIS

As the most common form of tuberculosis in adults, and the chief source of all forms of tuberculosis in man, pulmonary tuberculosis looms large in medical practice; and the skilful physician has the deepest interest in all that helps in the early diagnosis of this all too common disease in its infinite variety and vicissitudes. It is not easy to become a faithful artist, and even the best artists can achieve great success only by dint of infinite labour. Early diagnosis is the secret key to successful treatment, no matter what principles of treatment may be adopted, and the physician who has learnt to recognise the disease in its earliest stage is the consummate artist. Once the physician has mastered the difficulties that beset him in his efforts to detect the disease at its very beginning he is master of the situation. The transcendent advantage of early diagnosis is one of the natural fruits of specific treatment. Until Koch had proved that pulmonary tuberculosis in the early stage could be "cured with certainty by tuberculin," early diagnosis had not the same significance. With specific measures early diag-

nosis is tantamount to success in treatment. If it be a paradox to say that prevention is the best treatment, it is no paradox to say that the best opportunity for treatment has passed if the physician overlooks pulmonary tuberculosis in the early stages. Treatment in the early stage may fail; that may be the fault of the disease, not of the physician. Failure to detect the disease in the early stage is the fault of the physician. The true artist in diagnosis sets up for himself a high standard. In his great task he uses whatever methods are at his disposal, bearing in mind the serious fact that if he overlooks an early case he is in some measure responsible for the life of misery and suffering that are the constant features of the later stages of this insidious disease. Failing to recognise the disease he has failed in his duty to his patient and done him an irreparable wrong. The wound which the disease is thus allowed to inflict is truly a wound for which the physician must blame himself.

In every tuberculous process two factors are concerned; the specific extraneous and essential cause—the tubercle bacillus—and the environment, which embraces both the tissues themselves and conditions modifying them. In the early stages of pulmonary tuberculosis we may assume that in most cases the tissues themselves are sound and healthy till the tubercle bacillus enters. In other cases the soil is prepared for infection. Thus measles and whooping-cough dispose to tuberculous disease. This disposition was well illustrated in a case of pulmonary tuberculosis that was being treated with tuberculin. The patient made good progress, and at no time were the reactions anything but mild. During the course with tuberculin she had a severe attack of measles. On resuming the treatment after the attack of measles she exhibited extraordinary susceptibility to tuberculin. In some way the attack of measles rendered the system far more sensitive to tuberculin. If we consider the disease to be merely the expression of an affinity of the tissues for the bacterial toxins we learn that one infection may render tissue more sensitive to another infection. It is well known that influenza may increase the susceptibility to tuberculosis, and I have observed that even severe colds increase the

susceptibility to tuberculin. In this way influenza, or a severe chill or cold, may light up a latent tuberculous focus by increasing the disposition to the tuberculous process. So, too, other infections due to streptococci, pneumococci, staphylococci, etc., may prepare the soil for the tubercle bacilli. Thus the ætiological diagnosis must take into account other infections, especially the common infections of the air passages. These infections favour tuberculosis, and tuberculosis favours these infections. This vicious circle of tuberculosis and secondary or concurrent infections may play an important part even in the early stage of pulmonary tuberculosis, though, as a rule, these unfortunate accidental complications belong to a later stage of the disease. Still it is no unusual experience to find infection with streptococci or pneumococci or influenza bacilli complicating the early and curable stage of the disease, so that an ætiological diagnosis at the very earliest stage of the disease demands a careful and systematic analysis of the discharges from the air passages, whether mucus or pus or blood in varying proportions, by all the resources of bacteriological science. Whenever there is sputum a bacteriological diagnosis is indispensable. It goes without saying that the absence of tubercle bacilli from the sputum in no way excludes tuberculosis. Certainly the newer methods of searching for tubercle bacilli in the sputum by means of anti-formin enables us to detect tubercle bacilli in the sputum even when they are very few in number. On the other hand, we must remember that there are forms of virulent tubercle bacilli which are not stained by Ziehl's method; some tubercle bacilli, in fact, are not acid-fast. It is even said that in 12½ per cent. of these cases this form appears. Then it is necessary to use Much's modification of Gram's method when tubercle bacilli appear in the granular form, sometimes resembling streptococci, though much smaller in size; but in 90 per cent. of pulmonary tuberculosis in the early stage, tubercle bacilli are not to be detected, even with these improved methods, because the tubercle bacilli cannot escape from the tissues which imprison them. I fear that occasionally physicians are rash enough to pronounce an opinion without even examining the sputum;

while others are too fond of pronouncing against tuberculosis because tubercle bacilli have not been discovered. Both errors may end in disaster to the patient. If after two or three careful examinations no tubercle bacilli can be found, judgment must be suspended until the acute stage is over. Then when the temperature has become normal the tuberculin test should be exploited; otherwise errors in diagnosis are inevitable. Too often the patient is allowed to live in a fool's paradise, for even the disappearance of fever and abatement of all symptoms do not justify the view that there is no tuberculosis. Ofttimes the disease is called influenza—a convenient term for the careless physician—or what you will, and months or years after, when the disease has gathered strength, the patient has no recollection of the warning, alas! disregarded by his physician, and the physician himself will scarcely have the hardihood to acknowledge his disastrous and maybe fatal mistake: "Where ignorance is bliss, 'tis folly to be wise." Such mistakes are very common, as I know by experience, though often patient and physician alike remain in a blissful ignorance that causes no remorse. It may happen also that, when the advice of the physician is first sought, the disease has already advanced to a serious stage. I have seen perhaps half a dozen cases in which no suspicion of serious trouble had been raised, although the first examination revealed disease passing into the third stage. In such cases early diagnosis is impossible unless the family physician keeps a vigilant eye upon each member of the family he attends, especially if there is any cough or loss of health, strength or weight. The family physician will find abundant opportunities for exercising his judgment and authority in this way, and whenever there is a shadow of doubt the wise man will play trumps and seek out the physician, who will leave no stone unturned till he can say to the patient Yes or No. The yes-no diagnosis in pulmonary tuberculosis is unworthy of the medical science of to-day. How often, alas, the family physician affects to know before he has gone to the trouble of satisfying himself that there is no treacherous enemy lying in ambush.

Beyond all doubt the early diagnosis of pulmonary tuber-

culosis was extremely difficult—indeed, often well-nigh impossible—in the days when implicit trust was placed in physical signs and symptoms. Mistakes were many and inevitable. Physical signs may mislead, for the signs of localised catarrh due to various infections may simulate the signs of early tuberculosis, even at the apex, and, on the other hand, the early symptoms of pulmonary tuberculosis may have little or no relation to the lungs. Two kinds of mistakes are possible. Cases of true tuberculosis of the lungs may be overlooked, and, perhaps the more common mistake, many cases that are not tuberculous may be treated as such, thus swelling the lists of successes by various methods of treatment. Even in his latest report (1904) Weicker tells us that among the applicants recommended for his sanatorium by physicians well versed in the older methods no less than 48 were rejected as non-tuberculous. Koch's great discovery in 1882 soon established the presence of tubercle bacilli in the sputum as the test of tests. For many years this test by means of special stains dominated everything, but deeper knowledge shows that this test fails when it is most wanted.

It fails and misleads in those early stages of tuberculosis of the lungs in which the tubercle bacilli have not escaped into the sputum. The extent of this error may be gauged by the simple fact that in the best sanatoria the selection of early cases is so exclusive that not one-third of the accepted cases have tubercle bacilli in the sputum. The presence of tubercle bacilli in the sputum is the most positive evidence, but the absence of tubercle bacilli from the sputum does not exclude tuberculosis. The records of sanatoria show that even in the second and third stages tubercle bacilli may not be found in the sputum. Thus, nowadays, the test is neither invaluable nor indispensable, and as a rule the presence of tubercle bacilli in the sputum is in itself a sign of rather a late stage, for disintegration of tissue must have occurred, and if disintegration has occurred physical signs are to be found, because in the vast majority of cases the earliest lesions occur at the apex, which is within reach of percussion and auscultation. In rare cases a primary central lesion may not yield definite physical signs. Thus, staining sputum for tubercle

bacilli has lost its importance in early diagnosis, since the aim of the consummate artist in diagnosis is to detect pulmonary tuberculosis long before tubercle bacilli have escaped into the sputum. If we measure a man's progress by the size of his scrap-heap we may say almost that the staining of sputum for tubercle bacilli for the diagnosis of early tuberculosis of the lungs must be consigned to the scrap-heap. The new doctrine that successful treatment ends where physical signs begin has at least this virtue, that it emphasises the value of early diagnosis. Nevertheless tubercle bacilli may occur in the sputum in an early stage, and sometimes it happens that tubercle bacilli are found when the physical signs are trifling or negative; but particularly in cases of bronchitis, pneumonia, influenza, and pneumo-coniiosis, the discovery of tubercle bacilli in the sputum is of great significance, though for a time the tubercle bacilli may play a subordinate rôle. We may err by giving too much prominence to the tubercle bacilli. A tuberculous lung enjoys no immunity from other infections, and, should acute infections with influenza bacilli, streptococci, or pneumococci supervene, we may greatly err in diagnosis if we call the disease acute tuberculosis when after all the acute condition is independent of tubercle bacilli. The discovery of tubercle bacilli in the sputum is specially important in mixed infections and when physical signs are doubtful or absent. Let me add, too, from an extensive experience that the examination for tubercle bacilli in the sputum is not always a simple matter. Often enough they are readily found in properly made specimens, but a negative result, except in the hands of an expert, is almost worthless. Special methods may be necessary to bring the bacilli into view or prove their presence. But even when we fail to demonstrate tubercle bacilli in the sputum our resources are by no means exhausted, and the full truth of this fact is slowly dawning upon those who have long resisted the clearest indications of progressive measures and recent research. When tubercle bacilli fail in the sputum definite symptoms may fail; physical signs may help or may be on the border line of variations consistent with health. If physical signs be present they may be merely the inevitable signs of an obsolete tuberculous focus or the result of those

defects in development of certain areas of the lung to which Hirsch-Hirschfeld and others have directed attention, or merely the signs of various infections that cause localised catarrh of the bronchial tubes, or even the signs of a normal lung wrongly interpreted. On the other hand, in the early stages of pulmonary tuberculosis neither physical signs nor symptoms may avail us in diagnosis, and the real nature of the disease is easily and, as I know, often overlooked. It happens too often that the disease is recognised for the first time when it has passed into the second stage under the very eyes of the physician; or the truth may be forced upon him with a shock when a severe hæmorrhage unmasks the disease even to the layman, or an acute secondary infection brings tubercle bacilli into the sputum or causes the classical symptoms of true phthisis—fever, night-sweats, wasting, etc. The physician plays his part best who anticipates such catastrophes and does not fail to seize the golden opportunity for treatment, which may come once never to return. The great lesson of the last decade is the paramount advantage of detecting and treating pulmonary tuberculosis in the early stage before tubercle bacilli reach the sputum, while in fact the tuberculous lesion is still closed, or, in other words, shut off from the external world represented by the air passages. If at this time symptoms are vague and misleading, if signs may fail us, if tubercle bacilli are still imprisoned in the tissues and cannot escape into the sputum, how may we, in spite of the difficulties, recognise a disease that thus parades itself in the garb and trappings of health? The genius of Koch has furnished a simple and safe solution to our otherwise dire perplexity. In November, 1890, over 19 years ago, Koch wrote: "I believe I am not going too far in assuming that tuberculin will for the future constitute an indispensable agent in diagnosis. By means of it one will be in a position to diagnose doubtful cases of incipient phthisis even when, on account of the absence of bacilli or elastic fibres, or regular physical signs, we cannot obtain certain information concerning the nature of the lesion." Whatever ills and disasters have been laid to the charge of tuberculin by the insensate fears of the timorous, the absurd reasoning of the ignorant, or the distorted imagination of the sceptical wiseacres,

those who have constantly used tuberculin both for diagnosis and treatment, and therefore have established some claim to be heard, agree with the great master that tuberculin is the invaluable and indispensable key to the early diagnosis of pulmonary tuberculosis. Tuberculin rarely deceives the expert and never does any harm in the expert's hands. Tuberculin is at once a simple, safe, and trustworthy agent in diagnosis, powerful for good, powerless to do harm, if its use be rightly understood. It is to be deplored that many lives have been sacrificed to a strange and surprising disregard of this transcendent discovery, and it is high time that the systematic use of tuberculin should be substituted for the haphazard methods that often allow the victim to drift into that stage when recovery is difficult and tedious. In the early stage, which tuberculin reveals with such certainty, tuberculin is the remedy *par excellence*. By the use of tuberculin as a curative agent the demands of hygiene are likewise satisfied, since every case thus cured is one case less to form a centre and source of further infection. Etiological diagnosis must also take into account the soil as a factor in the disease. The bacillus is not the disease, even when it is in the tissues, nor yet is predisposition a state of disease, though Prof. Behring would have it so. The disease is the result of the affinity between the bacillus and the tissues.

A few words about the strains of tubercle bacilli that may infect man will not be out of place. The researches of Kossel, Weber, and Hesse prove that two distinct groups of tubercle bacilli occur in warm-blooded animals. One strain causes tuberculosis in the bird tribe, the other strain occurs in two distinct types—the human and bovine. The human type is the common cause of tuberculosis in man; the bovine type causes tuberculosis in bovines and swine. Nevertheless the bovine type occasionally causes tuberculosis in man, almost always, if not exclusively, in children; but even in children the usual cause of tuberculosis is the tubercle bacillus of the human type. In Kossel's investigation the bovine type was not even once found to be associated with pulmonary tuberculosis in the adult. Recently Kitasato has reported to the congress at Buda-Pesth that in 132 cases of pulmonary tuberculosis the

tubercle bacillus was obtained directly from the sputum, and subsequent investigations proved that these tubercle bacilli, without a solitary exception to prove the rule, were invariably of the human type. Accordingly, man is the all-important source of tuberculosis in man. Yet, since young children may be infected with bovine bacilli, cow's milk may be a source of danger. It is quite possible that in the near future we shall be able to differentiate between the tuberculosis of bovine origin and that of human origin in human beings. Such a diagnosis may be possible when we have studied more closely the reactions of tuberculin of various origins. Meanwhile we may rest satisfied with the now generally accepted doctrine of Koch, that the old, much-abused tuberculin of Koch is the most trustworthy test of tuberculosis in man. Few will attempt nowadays to dispute that, in doubtful cases of tuberculosis, when clinical evidence and physical signs leave us in the lurch, the negative reaction, obtained according to the conditions expressly laid down for us by Koch in 1890, excludes with certainty the existence of a tuberculous lesion. Thus, in early suspected cases a negative reaction with tuberculin, properly used, is positive proof that there is no tuberculosis. By this negative test I have been able to prove that a wrong diagnosis has been made by numbers of medical men, and I have thus saved many persons the expense and annoyance of exile to the country for the purpose of curing a disease they had not got. Such mistakes in diagnosis swell the successes of open-air treatment. Fortunately, these mistakes are rare at the best sanatoria, because tuberculin is used as a matter of routine in these doubtful cases. Unfortunately, the medical profession in England still looks askance at tuberculin, and wilfully blunders. If an active tuberculous lesion exists, the reaction will occur unless the living tissues have been immunised against tuberculin, either by artificial means or by virtue of a slow process of active immunisation during the progress of the disease. In the latter case there will be abundant evidence of tuberculous lesions, as this immunity develops only in old standing cases, so that, under these conditions, a negative reaction should not mislead. Everyone who has had an intimate and extensive acquaintance with the

effects of tuberculin in the various stages of pulmonary tuberculosis knows that if tubercle bacilli are found in the sputum a negative reaction with tuberculin practically never occurs. I can confidently assert that in my experience with tuberculin of all forms for purposes of treatment I have not met with a case of pulmonary tuberculosis, exhibiting tubercle bacilli in the sputum, in which the tuberculin reaction did not occur; in other words, a positive reaction always occurs when tubercle bacilli are present in the sputum. Thus, under the severest form of control, we can show that tuberculin never fails as a test for pulmonary tuberculosis. It surely cannot be argued that the escape of tubercle bacilli from the body helps the tuberculin reaction to assert itself. There is still the question whether a positive reaction with tuberculin may occur when no tuberculosis exists. If a positive reaction implies not merely a definite rise of temperature, but also a local reaction at the seat of the lesion, the inference is obvious. Thus if after an injection of tuberculin there are evident rales at the apex in the suspected area, if the cough is increased and the sputum also increased, maybe tubercle bacilli are then found for the first time. If there is pain and swelling in the glands, joints, or skin, none but the blind can mistake the signal. Moreover, in suspected renal or vesical tuberculosis, in ovarian or tubal tuberculosis, the local reaction also tells the tale. There may, indeed, be a marked local reaction without much rise of temperature, say above 100° , when hitherto the temperature has been normal. Such a temperature, accompanied by a local reaction, points to tuberculosis. Further, it may be surely argued that if there is a positive reaction of fever (100° , 101° , 102° , etc.) without definite local signs or symptoms, and subsequent treatment with tuberculin causes general improvement of health, increase of weight, improved appetite, restoration of energy, and abatement of such symptoms as cough and expectoration, there can be little doubt that a tuberculous lesion exists or has existed somewhere. Tuberculin can have no virtue unless there is tuberculosis. I have seen several cases in which, after a positive reaction of fever, the subsequent use of tuberculin for curative purposes has been followed by remarkable improve-

ment of the general health, although no definite local reaction occurred to indicate the actual seat of the lesion. But occasionally a positive reaction may mislead. Tuberculin may be too delicate a test. It may indicate a tuberculous lesion that exists and divert attention from even worse conditions. Such mistakes may occur when cancer attacks one already infected with tuberculosis. I have seen also a case of cancer in a young man of 29 which closely simulated tuberculosis. The patient gave a history of a diagnosis of abdominal hydatid, and there was a scar above the umbilicus suggesting the track of an abscess. I repudiated the diagnosis of hydatid and thought of tuberculous peritonitis with secondary invasion of the lungs. The patient was wasted, had irregular fever, and fullness and resistance in epigastrium; the signs in the right lung were those of disseminated miliary tuberculosis. He had sputum which, though repeatedly examined, yielded no tubercle bacilli. The tuberculin test could not be applied on account of the fever. He rapidly lost strength and wasted, and the necropsy disclosed cancer of the stomach and extensive disseminated miliary cancer of the right lung. Hundreds of miliary deposits of cancer not much larger than the ordinary miliary nodule of tuberculosis were scattered through the lung. The youth's age was the stumbling-block in the way of the diagnosis of cancer. In adult life there is rather the risk that, although tuberculosis exists and is recognised by tuberculin, worse conditions may remain undetected. There is no virtue in a tuberculous lesion, and its power for evil may be easily underestimated. Certain forms of acute tuberculosis are the direct result of the escape of tubercle bacilli from these quiescent lesions into the blood-vessels, sometimes, perhaps, via the lymphatics. I have seen several acute cases apparently of this nature, and until we take a serious view of the existence of even mild or quiescent lesions possessing unknown potentiality, and revealed to us by the tuberculin reaction, we shall be playing the brainless part of idle onlookers when we should be giving the enemy no quarter, and these acute cases will occur to our reproach and disgrace.

Even if but *one* quiescent focus in every four develops, he

who regards every tuberculous lesion seriously and treats it accordingly may claim 25 per cent. better results than he who, in a blind confidence, allows one case in four to assume a serious form. It is folly to underrate the enemy's strength, and in my experience it is not easy to overrate the treacherous ways of tuberculosis. Knopf's view upon tuberculin as a diagnostic test is certainly extraordinary, and I see no sense in it. He thinks that tuberculin should not be used as a test if it seems to do harm in one case in a thousand. It will, I presume, be conceded that harm can only follow where there is tuberculosis. The idea that a few doses of tuberculin, that cause no symptoms, can do harm is a virtual "Aunt Sally"—a poor sort of bait that is hardly worth the trouble of attacking and demolishing. All who have used tuberculin on a large scale know that in proper doses it can do no healthy person any harm. On the other hand, the early diagnosis, which gives the best opportunity for prompt and thorough treatment, is itself such an immense advantage that it is well worth the hypothetical risk of a little harm once in a thousand times. The great good of a certain diagnosis easily outweighs this hypothetical risk. I admit that the argument appeals with irresistible force to those who, having again and again proved that tuberculin treatment in the early stages immensely increases the percentage of permanent results can see no other course that appeals to reason than the adoption of all diagnostic measures that will bring under tuberculin treatment the very earliest stages of the disease. Every year fresh evidence strengthens the conviction that in the very earliest stages of pulmonary tuberculosis there is no other trustworthy test than tuberculin. The Arloing-Courmont agglutination test has been rejected by Koch, Behring, Fraenkel, Rabinowitch, Romburg, and others. I may add, too, that from my own personal experience with tuberculin as a diagnostic agent in many hundreds of cases I have not yet seen any harm result in those cases in which the test is necessary and justifiable. In the healthy, then, test doses of tuberculin do no harm; in cases of early tuberculosis the proper use of tuberculin can rarely, if ever, do harm; on the contrary, in my judgment, it always does good. I can further assert that, although I have

treated hundreds of cases of pulmonary tuberculosis in every stage with tuberculin in various doses, sometimes causing severe reaction, I have not once seen tuberculous meningitis supervene in any of the cases thus treated. As a complication of pulmonary tuberculosis, tuberculosis meningitis is not so very rare. In my hospital practice of the last four years I have seen several cases. Cases have been reported at the Wentworth Falls Sanatorium, which has only had a life of two years and is supposed to receive early and favourable cases of pulmonary tuberculosis. Yet in hundreds of my cases which have been treated with tuberculin, mostly with large doses, I have no single case of tuberculous meningitis to record—surely a noteworthy fact. I have come to the conclusion that, so far from tuberculin mobilising tubercle bacilli, tuberculin prevents mobilisation, and therefore prevents such accidents as tuberculous meningitis supervening in pulmonary tuberculosis. I leave it for those who do not use tuberculin to explain why they should conjure up for their own satisfaction those disastrous effects of tuberculin which do not occur in the experience of those who have used it. I can only assert that as the result of treating the first stage of pulmonary tuberculosis with proper doses of tuberculin, and it may be necessary to repeat a course, I have not yet seen a case in the first stage pass into the second stage. I knew of one or two patients who submitted to a partial course and were dissuaded by medical men from continuing the treatment, in spite of the fact that during the course the patients improved considerably. In these cases, thanks to the interference of other medical men, relapse, progress of the disease, and death have been the result. No such result has ever happened in an early case that has been submitted to proper treatment. I knew also a patient in the first stage who refused to be treated with tuberculin and preferred to go to the Wentworth Falls Sanatorium. After the sanatorium treatment he came to see me again, and I found that he was passing from the second stage into the third. This is not tantamount to saying that no cases in the first stage are arrested by sanatorium treatment. I know otherwise, just as I know that cases in the

first stage, even with tubercle bacilli in the sputum, will get well without sanatorium treatment and may certainly get well with it.

Thus ætiological diagnosis is the key to successful treatment, and it remains only to say that if the medical profession had the courage to exploit tuberculin in diagnosis and treatment we should be drawing near to the "consummation devoutly to be wished" when the dread disease, pulmonary tuberculosis, would be cured in its early stages, and the horror, suffering, and misery, nowadays associated with the second and third stages of this disease, would be largely mitigated. Since the year 1891 I have treated more than 120 cases of pulmonary tuberculosis in the first stage, not one of which has progressed to the second stage. According to averages 40 or 50 of these patients ought to be dead. They are alive and well and should remain so. Through my work also 120 sources of infection have been prevented. Thus the work of a unit has produced a perceptible effect upon the death-rate from tuberculosis. If 50 medical men working in the same way had produced the same results the death-rate would have fallen considerably. In this way the profession will attain to far better results than by means of sanatoria or health tracts. Sanatoria demand irksome conditions for the beauevillers, and tracts leave no lasting impression. The medical men of to-day have the opportunity to perceptibly reduce the death-rate from tuberculosis two or three years hence. Will they not think of the future generation in their enthusiasm for better things? If my argument be sound the prevention of consumption demands two conditions; firstly, that the first stage should be effectively cured, and, secondly, that the late stages should cease to be sources of infection. The first condition is fulfilled more or less thoroughly by the use of tuberculin in diagnosis and treatment. The second demands that government should introduce some effective scheme for dealing with these constant sources of infection. If these two conditions were faithfully fulfilled we should no longer need expensive methods, such as sanatoria, except for the cases that are not in the first stage nor yet in the third. Ultimately the idea

that sanatorium treatment can be a reasonable measure in prophylaxis must go to the scrap-heap, and the scientific discovery of Koch, neglected already for 15 years, will prove to be the greatest boon ever evolved by the scientific genius of man.

TUBERCULIN IN DIAGNOSIS.

Let us proceed to discuss in greater detail the evidence which justifies the conclusion that tuberculin used for diagnosis according to Koch's method (1) is perfectly safe, and (2) is a trustworthy test of the existence of a tuberculous lesion. If we can establish these propositions, tuberculin should be used many times every week in every large hospital—merely for purposes of diagnosis—so that the advantages of this simple, safe, and trustworthy means of diagnosis should be exploited to the full, in the interests of science and of humanity. Once tuberculin comes to be freely used in hospitals as a diagnostic agent it will gradually take its rightful place as the best weapon as yet discovered for the treatment and arrest of tuberculous disease—at any rate of pulmonary tuberculosis. Old Tuberculin is the preparation which Koch has recommended for diagnostic purposes, and since the year 1890 I have used this preparation in the manner prescribed by Koch on an extensive scale, and in my long experience I have not yet seen any effect that would lead me to think that these doses ever did harm. On the contrary, in suitable cases these test doses always seemed to do good. The patients voluntarily admit that after the doses they feel better, and almost always there is a gain in weight, lessening of cough, and improved appetite. Accordingly, after nine years' experience, in 1899 I wrote as follows:—

"Excellence of any kind—in anything—is not easy of attainment. The student of to-day, if he compares his knowledge and methods with the knowledge and methods of twenty years ago, may have some reason to be proud of his superiority, yet twenty years hence his knowledge and methods may be old and out of date. Neither finality nor perfection is reached in anything, and the open mind possesses the great advantage of being able to assimilate without any effort anything that

savours of truth, even though it be out of harmony with current ideas. At the same time circumspection is necessary, lest the receptive mind, untutored of experience, wastes its energy in fruitless chases after will-o'-the-wisps. Our Profession, if it has been famous in history for anything, has been famous for its strong tendency to a rigid conservatism. If any great teacher or reformer arises, his views, if in advance of professional opinion, are likely to meet with a more or less obdurate, unreasoning resistance. This attitude of antagonism to new ideas and discoveries is illustrated almost every day, perhaps because the study of medicine in the past has offered a fine field for reckless dogmatism and redundancy of wild theorising. History repeats itself. Hippocrates had to suffer the fury and indignation of his craft when he boldly divorced the art of medicine from priestcraft. When Harvey enlightened the whole world of science upon the nature and purpose of the heart and blood-vessels he lost most of his friends and his patients left him. Sydenham encountered the scorn of the Profession in his day, because he asserted that quinine, even though it did not purge or sweat the patient, and without even the help of the bleeder, was still a specific for ague. Sydenham was an unmitigated heretic because he conceived the idea that a drug might possess some virtue even though it did not sweat or purge. Moreover, in Sydenham's opinion quinine rendered the invariable bleeding a superfluous luxury. The bleeder only objected to this view, the bled had no choice in the matter. Lister was belittled and ridiculed by his own brethren when he was painfully and anxiously labouring with the embryo idea that was destined to revolutionise and dominate the whole field of surgery. It is so difficult to pick out the grain of truth in the bundle of chaff. Absolute truth, like perfection, is hardly attainable, and if truth is half hidden in error, the ordinary observer sees but the error. The mind seems to have a far keener eye for faults than for virtues. In my humble judgment the rejection of tuberculin as a diagnostic agent is a very striking illustration of this attitude of mind. Who amongst us, who among English physicians, who among the Germans themselves, except Koch's own pupils, is able at the present time to pronounce an opinion based on experience

on the value of tuberculin in the diagnosis of tuberculosis in human beings?"

By Koch's method the rise of temperature is the sign of signs. This cannot mislead as subjective symptoms may. The temperature may rise to 100°, 101°, 105°. The lower temperatures last a few hours; the higher temperatures last longer and may extend over two or three days. Afterwards the temperature returns to the normal. Even a small dose may cause a great rise of temperature, especially in lupus, and sometimes in early lesions. In 1890 I gave the first test dose to a man who had been two years in a country sanatorium. This man had been under my care in a hospital two years before. When I saw him at the sanatorium he volunteered the statement that I had pointed out to some medical students that the left apex was the seat of early trouble. Examining him again I found no very definite physical signs. He had been at the sanatorium for two years, and was there still, unfit to work. Had the rest and fine climate cured him? The tuberculin test told the true tale. With a dose of .001 cc. of Old Tuberculin he reacted to 104°. Thereupon I treated him with Old Tuberculin. In two months he gained a stone and was able to return to work. Eleven years later I heard of this man as being still well. This man had been at the sanatorium for two years, but was still ailing; after tuberculin treatment he was able to return to work and he was well eleven years later.

This first experience impressed me, as well it might, for it seemed to prove that—

- (1) Tuberculin was the only means of demonstrating the presence of a latent tuberculosis that was affecting the health.
- (2) Sanatorium treatment and open-air treatment tried for two long years had failed to restore health.
- (3) While sanatorium methods had utterly failed, tuberculin as a curative agent succeeded immediately it was tried, and in three months' time.
- (4) The tuberculin treatment secured a permanent result (eleven years).

Since 1890 I have used tuberculin for diagnostic purposes in many hundreds of cases and have never seen any harm from its use, and many times have I seen all the signs of improvement after the reaction is over.

Obviously one uses tuberculin most often in the diagnosis of chronic tuberculous diseases of the lungs, because in cases of fever tuberculin is not used for diagnosis—at least not until the temperature has returned to the normal level and remained there for a few days. In cases of bronchitis, asthma, bronchiectasis, hydatid disease of the lung, especially with hæmoptysis, tuberculin is invaluable in diagnosis. In most forms of pleurisy, especially in the less acute forms, but also in acute pleurisy after the fever has passed away, tuberculin surely proves the tuberculous nature of the condition. I have detected tuberculosis even when all the other symptoms disposed me to exclude it. In cases of bronchiectasis I proved by means of tuberculin that there was no tuberculosis. I have also tested several cases of laryngeal catarrh, and in some of them the reaction to tuberculin just shed that light upon the true condition that enabled one to treat with tuberculin the early stage, that might otherwise have passed into the late stages of pulmonary tuberculosis. In ulcers of the nose, of the tongue, the pharynx, and the palate I have used tuberculin in order to establish the diagnosis. In Australia lupus is a very rare disease, but I had occasion to diagnose it twice by means of tuberculin—in one case proving that lupus had engrafted itself upon a rodent ulcer; in the other case settling the diagnosis of a typical case of disseminated lupus, the subject of which ultimately died. I began treatment with tuberculin in the latter case, but after a few weeks the friends of the boy took him up the country, and unfortunately treatment ceased. In other skin lesions simulating lupus I have been able to exclude lupus. Also in cases of tuberculous peritonitis and (in women) of tuberculous salpingitis I have proved the tuberculous character of the condition.

In suspicious cases of chronic pyelitis or of renal trouble I have several times used the tuberculin test in diagnosis, and am astonished that so many writers in discussing the diagnosis of renal tuberculosis overlook the tuberculin test. Not only

is tuberculin of very great value in the early diagnosis of renal tuberculosis, which so often runs a latent course even for years, but renal tuberculosis in my experience seems to be particularly amenable to tuberculin treatment. Unfortunately, troubles of the bladder and kidney, especially associated with pyuria, are supposed to belong to the domain of surgery, and I have found that surgeons especially look askance at tuberculin, so that I have seldom had opportunities of showing the value or otherwise of the tuberculin test in the early stages of renal tuberculosis. This field is only waiting to be explored by the searchlight of tuberculin, and I believe a rich harvest of results will be reaped thereby. Now, if tuberculin is so valuable in these rarer manifestations of tuberculosis, it becomes tenfold more valuable in pulmonary tuberculosis, because this form is more than ten times as prevalent as the other forms; and in the gland tuberculosis of children, especially bronchial tuberculosis, as well as in tuberculosis of bones and joints, there is a wide field for the exploitation of this method of diagnosis.

Let us now briefly consider whether tuberculin as a diagnostic agent is quite safe.

One has to admit that there is a very strong prepossession in the minds of medical men that the test is not free from danger. The opposition of those who suppose that the injected fluid, which is always boiled, may convey tuberculosis is based on such ignorance that we may ignore it. It is also not possible to give any good reason why tuberculin should do any harm in a healthy person. I have given to myself test doses, and in the hospital and in private practice I have used the method in a routine fashion—not only scores, but hundreds of times—without seeing the least effect, either good or bad. Nor have I ever heard of tuberculin affecting in any way a healthy person. Accordingly we may confine our attention to the question whether tuberculin, used as a diagnostic agent in suitable cases according to Koch's directions and limitations, can do any harm in those affected with tuberculosis. It is, of course, impossible to assert or deny that tuberculin, used for diagnostic purposes, may apparently increase the activity or extent of an already existing tuber-

culous lesion. At any rate, it is certain that any harm happening can only occur in those who are already tuberculous. After an experience extending back for 20 years, during which I have freely and openly used this test, I am ready to solemnly affirm that I have not yet seen a serious effect from the use of tuberculin as a diagnostic agent. For the last eight years I have used tuberculin in routine fashion at a hospital where everyone used to be extremely hostile to this method, but now everyone is convinced, by the results in my wards, of its harmlessness.

It counts for something that, though my work in this direction in a public hospital has been closely watched, even by those who have no friendly feelings towards me, the only effect has been that the whole staff has been converted to the view that tuberculin used for diagnosis in the proper way does not work any harm. I am proud to be able to say that there is no hospital in the British Empire where the value of tuberculin in diagnosis is more thoroughly recognised. But though I have been fortunate enough to use tuberculin as a diagnostic agent in many hundreds of tuberculous persons without any harm or accident, I do not deny that apparent harm or an accident may possibly follow the use of a small dose of tuberculin, as it may follow a cough or a muscular effort, or a change to a high altitude. Two questions, then, present themselves: (1) How often does it follow such an injection? (2) How often is it due to the injection? In my experience it has not once followed the injection; and in the experience of others, if it has done so, it still remains to be shown that it was caused by the injection.

We are all familiar with the sudden accidents that can happen in England even in the early stages of pulmonary tuberculosis—a hæmorrhage—an acute pleurisy—a mixed infection—even a pneumothorax—or rarely a tuberculous meningitis, due to generalisation of the process. No one denies that these accidents occur without tuberculin. Who has dared to produce evidence that these accidents are more frequent when doses of tuberculin have been given? If any such evidence had been forthcoming, I should have produced very remarkable evidence to the contrary. If there is one

thing certain, as far as my experience goes, it is that tuberculin is far the best remedy for the arrest of hæmorrhage. Hæmorrhage is an indication for, not, as some would have it, a contra-indication against, the use of tuberculin. I have proved this in scores of cases, and am able to assert that after treatment with tuberculin hæmorrhage is a very rare accident. Hæmorrhage is a common accident in pulmonary tuberculosis. Hæmorrhage is rare in cases that have been treated by tuberculin, so that, far from tuberculin causing hæmorrhage, this remedy is the remedy *par excellence* for the arrest of hæmorrhage.

But although my experience gives no support to the popular view that test doses of tuberculin may not be free from risk, I am willing to argue that the risk is not great enough to justify the condemnation of the method. In many other conditions we have to take risks in order to make a diagnosis. We use the exploratory needle in the thorax, in the abdomen, and even in the spinal cord and brain. Have accidents never followed these trifling operations? The exploratory needle may leave you where you were. The injection of tuberculin leads you to certainty. And how supremely important it is to be certain and positive when one is consulted, and has to give an opinion, upon the state of health of a boy or a girl, or, more serious still, of a man in active employment or a woman with all the cares and duties of a household upon her shoulders. Some are ready enough with the advice to take a change and go into the country for several months. I have records in which patients on medical advice have given up good positions and left England for Australia. In one case the patient came to me within a week of his arrival in Australia and I tested him. He gave no reaction at all, nor was there any evidence of old or recent disease. If, indeed, there is tuberculosis, such great sacrifices must oftentimes be made (though not necessarily if tuberculin be used); but surely we should be certain of our diagnosis, if certainty be possible, before we advise such serious measures. Think, for instance, what a sacrifice it would mean for a medical or professional man! Is it right, is it honest, to suggest such sacrifices, unless we are certain? Nothing short of certainty should

determine us in our duty, when certainty can be attained at so small a cost.

If the patient reacts to tuberculin, the sacrifice may be necessary, though I hold that many cases of pulmonary tuberculosis can be successfully treated with tuberculin without loss of employment or occupation. If, however—as too often happens—say in early and obscure cases of laryngeal or pulmonary tuberculosis, the test is not used and the diagnosis is held in abeyance or, perhaps, an absolutely wrong diagnosis is made, the disease may soon pass beyond that stage when treatment is most hopeful. That injury to the patient can never be undone; and I am bold enough to say that the injury that comes of not using tuberculin as a test, or failure of diagnosis because its use is withheld, far outweighs any injury that is likely to occur from its use in expert hands. I have many records in which throat specialists have not recognised tuberculosis, and within two years the patients have passed into the third stage with tuberculous disease of the larynx. In laryngeal tuberculosis the diagnosis by sight cannot be certain until typical infiltration or ulceration has occurred. With the help of tuberculin the diagnosis may be rendered certain months before these serious lesions develop, and by specific treatment ulceration may be prevented. Both in laryngeal and in pulmonary tuberculosis the earlier the treatment the better, and the tuberculin test ensures the very earliest treatment.

On the other hand, if the patient does not react what a still greater point of vantage has been gained—at no cost at all. One can assure the anxious patient that, whatever other trouble he may have, he has not tuberculosis. The reaction occurs only in tuberculosis, and in my opinion the reaction is well worth suffering to gain the immense advantage of an early and certain diagnosis.

I imagine that, just as I have many times found the test invaluable and indispensable in ear, nose, throat, and chest work, others would find it equally useful in tuberculous lesions of other parts. Does the gynaecologist never suspect tuberculosis of the tubes, or ovaries, or peritoneum, or even of the uterus itself? Does the oculist never think of an incipient

tuberculosis of the iris or conjunctiva? What is phlyctenula? Is it ever tubercular? Is a surgeon never in doubt about the presence of tubercular disease in a bone, or joint, or gland? May not even the physician call tuberculin to his aid, apart from cases of pulmonary tuberculosis? Tubercular glands of the neck may simulate lymphadenoma.

An inquiring physician may some day discover that ulceration of the stomach may be sometimes tubercular. Gastritis, gastric ulcer, anemia, chlorosis, and even rheumatism are all names under which tuberculosis may masquerade before the mental eye of the physician who forbears to use tuberculin. Appendicitis may have relations with tuberculosis. Women especially are accustomed to swallow their sputum, and the infective tubercle bacillus may invade the stomach in the absence of the acid gastric juice. It may pass into the intestines and lodge in Peyer's patches, or even in the appendix. Until this scientific test is generally used, we must remain in the dark concerning many doubtful and obscure points in the aetiology of disease. Many important lessons about tuberculosis remain to be learnt as soon as the tuberculin test is used as the key.

The systematic use of tuberculin as a diagnostic test can alone determine the prevalence of tuberculosis in human beings. We know what startling discoveries followed the extensive use of tuberculin as a test for tuberculosis in cattle. Before tuberculin was discovered the prevalence of tuberculosis in cattle was not known. At slaughter-houses where animals were subjected to some sort of inspection, the condemned animals did not amount to 5 per cent., and it was not supposed that tuberculosis existed even to the extent of 5 per cent., in cattle. As inspections became more careful, and inspectors better trained, the percentage rose from 5 per cent. to 10 per cent., and even 15 per cent. As soon as tuberculin was used as a test the percentage of tubercular animals rose to 30 per cent., 40 per cent., and even higher. These results were so startling and unexpected that no time was lost in casting discredit upon the test, and one of the highest English authorities asserted that the tuberculin test was fallacious in 25 to 30 per cent. of the cases. If this great English expert

had been right we must have said good-bye for ever to the tuberculin test. But this English authority found himself at variance with every skilled observer in France, Germany, Holland, Italy, and America, who without exception, bore witness to the remarkable trustworthiness of this scientific test. In his next report the English expert admitted his blunder. The most careful investigations in animals have been made with the tuberculin test, and controlled by the subsequent post-mortem examination of the animal, and at most a margin of error of 2 to 3 per cent. is granted; yet, when one fails to find a tubercular lesion after a positive reaction with tuberculin, it is still not merely possible, but probable, that the lesion is there, but has escaped detection. The tuberculin test is so delicate that it may succeed when both the eye and the microscope fail. The tuberculin test is, in fact, a safer guide than the eye or the microscope in an obscure case.

If the tuberculin test were generally used in human beings for purposes of diagnosis, the results might astonish us. Nowhere, so far as I know, has the tuberculin test been used on a large scale in human beings except in Koch's Institute in Berlin. In Koch's Institute the tuberculin test has been used in many thousands of cases. Very many of these were doubtful or suspicious cases of early lung tuberculosis, especially catarrh of the apex of one or both lungs. Of course, no tubercle bacilli could be found in the sputum. With tubercle bacilli in the sputum the case would no longer be doubtful, and the tuberculin test would be unnecessary. Of 338 doubtful cases injected with tuberculin, 298 (85.2 per cent.) reacted, and 40 gave no reaction. In tuberculous disease of the intestine and genito-urinary tract the reaction occurs just as promptly. Of 25 cases of suspicious ulceration of the larynx, 17 reacted. The co-existence of influenza and tuberculosis of the lungs was proved to be very common. Influenza is favoured by disease of the lungs, especially tuberculosis, and influenza works very unfavourably on the tuberculous process. In the severe epidemic of influenza in 1892 the high mortality from tuberculous disease of the lungs was a significant feature. Influenza complicating tuberculosis of the

lungs is a very dangerous mixed infection. In Koch's Institute out of 106 cases of influenza 67 reacted (63·2 per cent.); of 68 cases of pleurisy, 50 reacted (73 per cent.); of 66 cases of bronchitis, 29 reacted (43·9 per cent.). We have yet to learn how often bronchitis—especially the persistent bronchitis of advanced life—is tuberculous. I have seen typical examples of this error. Out of 76 cases of acute pneumonia tested in the convalescent stage 27 reacted, while only 4 out of 14 cases of emphysema reacted to the test. Of 17 cases of enlarged glands 16 reacted, and the odd case was shown, by an examination of an excised portion of the gland, to be a sarcoma. Of the cases of adenoids in children all but one reacted. This would almost suggest that adenoids were a tubercular condition. More than 50 per cent. of the cases of facial erysipelas reacted. Erysipelas of the face is commonly secondary to some septic condition of the nose, and in this tuberculosis may play a part. Of 25 cases of erysipelas in other parts of the body only 7 reacted. Reactions were also observed in septic cases, such as puerperal fever, after the fever had subsided. Reactions were very frequent in young chlorotic girls. Before tuberculin was discovered the relation between anaemia and tuberculosis had been established. Anaemia may be an early symptom of tuberculosis, but we do not know how or when a localised tuberculosis of the lungs or of a gland produces the anaemia. Probably the anaemia is due to a defect in assimilative energy in the tissues and blood-cells. The chlorosis favours tuberculosis, and tuberculosis increases the chlorosis. The primary fault in the tissue-cell favours both chlorosis and tuberculosis. Chlorosis also greatly favours chronic ulcer of the stomach, and some cases of chronic ulcer of the stomach may be tuberculous. It is probable that this ulcer is really due to a bacterial necrosis induced by changes in the gastric secretion, and there is no reason for denying to the tubercle bacillus this power of inducing necrosis.

Generally the stomach enjoys immunity from tuberculosis, perhaps by reason of its acid secretion; but the acid secretion may fail, and if tubercle bacilli enter the stomach with milk or butter, they may penetrate the wall and cause necrosis.

Subsequently the bacilli may be killed. At any rate, certain cases of ulcer of the stomach are on record which yielded to no treatment till tuberculin had been used. I have also recorded several cases in which gastric ulcer was the diagnosis, yet a reaction to tuberculin occurred and great improvement followed tuberculin treatment. In these cases the tuberculin produced a local reaction in the stomach, increasing the pain, and raised the temperature; and continued treatment with tuberculin cured the patients of a condition from which they had been suffering for years. If, then, in cases of chlorosis tuberculin has a similar effect, we may suppose that chlorosis is due to the tuberculoæ.

But in Koch's Institute reactions were also frequent in rheumatic conditions and infectious disease of the genital tract. Of 106 cases of gonorrhœa, 59 reacted, i.e. 55·6 per cent. Of 143 cases of syphilis, 59 reacted; 50 per cent. of the cases of gastritis reacted; 66·6 per cent. of the cases of gastric ulcer; and 53 per cent. of the cases of appendicitis, especially in women. Appendicitis may sometimes be of tubercular origin; and perhaps women, who usually swallow their sputum, suffer from this form.

Thus we find that in Koch's Institute the tuberculin test has been used in every form of disease, and the reactions have been obtained in an unexpectedly large proportion. In one series of observations 2,308 were injected with tuberculin; of these 1,525 reacted. Setting aside some obvious cases, in which the tuberculin test was not necessary for diagnosis, there remained 1,454 in which the diagnosis could only have been made by means of tuberculin.

A tuberculous lesion was shown to exist in more than 50 per cent. of the patients suffering from various diseases. This is, of course, but a starting-point. It will require years of close and careful observations to prove the value of these reactions. I need not add that these reactions were obtained in Berlin, where the most careful examinations led to the conclusion that one-third of the deaths of the manhood of the city is due to lung-tuberculosis. Many of these tuberculous lesions may be small, and may heal spontaneously; others may remain latent, but still be possessed of a self-contained

potency to grow and multiply, when occasion offers, to the injury and destruction of tissue, and, maybe, of life itself. We cannot guess what the systematic use of tuberculin in diagnosis may teach us. In any case, we have a definite starting-point, and the field before us is a wide and unexplored one. It is in these cases especially that Koch has claimed to have obtained such good results with tuberculin; but, in order that one may prove the value of tuberculin as a remedy for tuberculosis, one must persistently use tuberculin as a diagnostic agent, so as to detect the tuberculosis in the lungs when the stage is eminently favorable for specific treatment. In such cases tuberculin is the agent for diagnosis and the remedy for treatment.

I do not suppose for a moment that if we used tuberculin in this way, we should find latent tuberculosis as frequent as it is in Berlin, but I maintain that we should use it so that we may determine its prevalence, and especially that we may be able to detect the disease—commonly in the lungs—in the earliest stage. Beyond a doubt tuberculin should be systematically used as a test in all doubtful or suspicious cases of lung disease. Why should we hesitate? If the test is negative you have a right to assume that whatever other condition is present, tuberculosis is certainly absent. What comfort and peace of mind this certain knowledge brings to the hitherto anxious and careworn patient! Besides, he is spared the necessity that otherwise exists of spending a lonely existence in the country for the purpose of curing a disease he has not got.

Not very long ago I used the test in a man who had symptoms and signs simulating those of consumption, but his sputum showed no tubercle bacilli. I injected tuberculin with a negative result. Now, this man was a dairyman. Think what the value of the test was to him. He had been ordered by other medical men to give up his work and go up the country for some months.

Nor should we forget those mixed lesions of which one may be tuberculous. Some time ago I saw an interesting case of a mixed lesion. At first sight it appeared to me to be rodent ulcer. There was a deep excavation, with a raw sur-

face and a typical rolled-in edge. Still, one edge of the ulcer was less typical. I injected Old Tuberculin, and not only did the temperature rise to 103°, but the untypical edge swelled and became red, and treatment with New Tuberculin caused definite retrogressive changes in this part. The part became pale and dry. The large surface of the ulcer remained unaffected. Mixed lesions of tuberculosis and syphilis may also occur.

The uses of tuberculin may extend even further. We know that consumption is an infectious disease, and that usually infection occurs in the home, among those who are on terms of the closest intimacy. Indeed, this deadly enemy often finds in the very bonds of love and natural affection the best opportunity of showing its malignity. Thus it has happened that a son has infected a daughter, and a daughter perhaps a mother, before measures of prevention have been thought of or adopted. It is very important to determine whether the infection has gone still further. Tuberculin settles this point at once, and with absolute accuracy. A few injections will pick out the infected ones in the family, if any exist; and, again, treatment can be adopted at the most favourable time. Does not this represent an enormous gain? It matters not at all what ideas of treatment I or anyone else may have—whether we advocate climate or open air, or diet, or drugs, or massage, or hydropathy, or specific treatment, or combinations of one or more of these—however much we may disagree upon the kind of treatment, we all agree in this, that treatment by any method promises the best results in the early stages of the disease. With every kind of treatment the earlier the stage of tuberculosis the greater the chance of success. Is it not, then, passing strange that this tuberculin test, that alone enables us to make a diagnosis in the earliest stage of all, is, one may say, ignored? Birch-Hirschfeld's beautiful observations show that there may be multiple lesions in the bronchial mucous membrane that cannot give rise to any of the physical signs which we associate with the early stage of phthisis. Moreover, equivocal signs such as are commonly associated with early pulmonary tuberculosis may be due to the defective and arrested growth of certain

sections of the bronchial tubes, independent altogether of any tuberculous lesion. Tuberculin alone points out the truth.

In Sidney Martin's article in *Albhall's System of Medicine*, although certain difficulties in diagnosis are considered, tuberculin as a diagnostic agent is not even mentioned. In many of these doubtful or suspicious cases tuberculin would be of great value. In other cases, however, the presence of fever would *ipse facto* prevent the application of the test; but fever is by no means a constant symptom of tuberculosis of the lungs in the early stage. Anyone who keeps careful temperature records in cases of tuberculosis of the lungs must be struck by the frequent absence of fever. The temperature may reach 99°, but such a temperature does not contra-indicate the use of tuberculin as a test. In the meninges any increase of inflammation may not improve the patient, but in the many fever-free cases of tuberculosis of the lungs, of the larynx, in the nose, on the skin, on the tongue, on the pharynx, in the Fallopian tubes, in the pleura or peritoneum, in the genito-urinary tract, in the lymphatic glands, in the joints or bones, indeed in any situation, it surely is of the greatest importance to make an early diagnosis, and nothing but tuberculin can reveal the truth. It is idle to imagine that the first beginnings of pulmonary tuberculosis, when there may be at most a few scattered fresh tubercles, can be detected by any symptoms or physical signs, however skilful and experienced the physician may be. That a typical reaction to tuberculin in proper doses, including not only a rise in temperature but also a general disturbance and a very characteristic inflammatory lesion at the site of the injection, is a certain proof of the presence of tuberculosis is now generally admitted. The accuracy of the test has been abundantly demonstrated in animals in every civilised country. The thousands of observations in Berlin and other places prove that the test is equally trustworthy when it is applied to human beings. If, then, it is an unerring test, why is it not generally used?

Two chief reasons are urged against the general use of tuberculin in diagnosis:—

- (1) It is supposed that tuberculin may aggravate the condition.
- (2) Tuberculous lesions are so common in the healthy that a tuberculin reaction does not help the diagnosis.

(1) Let us consider the evidence favouring the idea that tuberculin may aggravate the disease. In the early stages of tuberculosis it has been my uniform experience that, while during the reaction and for three or four days after there is a loss of weight amounting to 1-3 pounds in proportion to the severity of the reaction, in the following three or four days the weight is not only restored to the amount before the dose was given, but another 1 lb or $1\frac{1}{2}$ lbs. is added. Thus within eight days there is a nett gain of $1-1\frac{1}{2}$ lbs in weight. Nevertheless it must be granted that aggravation of the condition did occur in many cases that were treated with tuberculin in the early history of tuberculin treatment. But these doses were not given in early stages—nor yet for diagnostic purposes.

This aggravation was due to the reckless use of tuberculin. Heedless of Koch's express limitations and restrictions, men were found ready and eager to inject tuberculin even into patients who at the best had but a few days to live. It will take years to undo the mischief of that reckless and insane misuse of a valuable remedy. Koch's warning was unheeded in the wild delirium of the moment. Koch stated that the remedy was especially valuable in the early stages of tuberculosis, and numerous witnesses vouch for the truth of Koch's statement that in such cases tuberculin is a most useful remedy. In early cases the benefit of tuberculin treatment had been demonstrated again and again; but in spite of treatment with Old Tuberculin, the disease may relapse. Because the remedy has fallen short of popular expectations, it has been derided and condemned as useless and harmful. What remedy that has ever been used fulfils the rigid conditions that some consider necessary before tuberculin should be allowed even a trial? What are our vaunted specifics? Do they never fail? Do we discard them if a relapse occurs? No remedy of the old régime holds a higher place than

quinine in the treatment of ague. Does quinine always cure? Do relapses never occur in the treatment of ague by quinine? We have yet to learn what rôle quinine has played in causing death by inducing hæmoglobinuric fever. Do we discard quinine because it has failed often, and caused many deaths? No. We learn to use it properly through the mistakes of the past. In spite of failures and fatal results, quinine is the remedy *par excellence* for ague. Why should tuberculin be discarded because it sometimes fails, or be condemned because in unsuitable cases it has hastened death? The pendulum will swing back in time, and tuberculin will find a place with us even as a curative agent. But why, because tuberculin fails sometimes as a curative agent, is it absolutely ignored as a diagnostic agent, although it never fails in this respect? Tuberculin ought to be used to establish the diagnosis, when all other methods fail. Then, and then only, can we expect to discover the disease in that early stage which yields most readily to suitable treatment. In man, as in animals—if the reaction is positive—it is a hundred to one that a tubercular focus exists somewhere, and often a local reaction will indicate at the same time the seat of the lesion. The tuberculin test will find its most useful application in early cases of lung diseases. Bösch-Hirschfeld has demonstrated these lesions to us in the dead body, and nothing can demonstrate their existence in the living body but tuberculin. But the tuberculous focus may be in the larynx, on the epiglottis, in the testicle, in the Fallopian tubes, in the bones or joints, and the local symptoms produced by the tuberculin may help us to locate the lesion. If, on the other hand, the reaction is negative, it is equally certain that no tubercular lesion exists. Certainly, in very advanced stages of lung tuberculosis, the reaction may fail because a certain degree of immunity to the effects of the toxin is reached; but in such cases the tuberculin test is no longer needed for diagnosis.

But the paramount question to be considered is the degree of risk that attaches to the use of tuberculin as a diagnostic agent in suitable cases. It may be taken for granted that the post-mortem examinations made by Professor Virchow on several cases of tuberculosis of the lungs that had been treated

with Old Tuberculin did much to raise the violent storm of opposition to the use of tuberculin under any circumstances. Professor Virchow and his school never had very much sympathy with the ideas and methods of Koch, and at this moment Professor Virchow's first lieutenant, Hansemann, is the leading representative of the school that denies the specific character of the diphtheria bacillus, and, moreover, is ready to criticise adversely the serum treatment of diphtheria. Yet Virchow's investigations constitute a most valuable contribution to the subject of the specific treatment of tuberculosis, and in very fact bear witness to the truth of Koch's own statements. They indicate in the clearest manner that there may be danger in using Old Tuberculin in advanced cases of tuberculosis, on account of the violence and extent of the reaction when the larger doses are used. But Virchow laid special emphasis on the opinion that tuberculin had a tendency to mobilise the tubercle bacilli—to break up the tubercular lesions, so that tubercle bacilli were loosened from the lesions in which they were embedded and carried into vessels. Thus it was imagined that tuberculin increased the tendency to generalisation of tubercle bacilli. Now, it is well known that, apart altogether from tuberculin, this generalisation of tubercle bacilli always takes place to some extent in advanced cases of pulmonary consumption. In these advanced forms of the disease localised lesions are common in distant organs—in the glands, in the kidneys, liver, spleen, and elsewhere—and tubercle bacilli are actually circulating in the blood. It is this positive condition that accounts for congenital tuberculosis. The tubercle bacilli are circulating in the mother's vessels, and pass to the placenta, and thence to the foetal vessels and tissues. We know, also, from observations in lupus, that tuberculin will bring into view nodules that were previously too small to be seen. It is, therefore, certain that after injections of tuberculin many microscopic nodules might be brought into view. But it would not be right to argue that the tuberculin had produced the new lesions. Hence, the presence of submiliary nodules around a tubercular focus after tuberculin, and even the existence of generalised tuberculosis, of greater or less extent, can be

explained without supposing that the tubercle bacilli had been mobilised. But even though some mobilisation may occur in advanced cases we have to consider the likelihood of such mobilisation merely in early cases, and that, too, when no more than one or two interjections are used. The changes observed by Virchow were found, without exception, in advanced cases that had been treated with tuberculin.

Now, what evidence is there that such generalisation may be brought about by the use of tuberculin in early cases, when it is chiefly used either for diagnosis or treatment? The best authorities are agreed that in animals, at any rate, there is no risk. Bang, who can base his opinion on 50,000 cases and more, says: "An acute exacerbation of tuberculosis after tuberculin injections is only to be feared as an exceptional occurrence, and that in cases in which an acute miliary tuberculosis not rarely occurs, even without tuberculin injections. With the great increase of tuberculin injections the number of cases of acute miliary tuberculosis ought to have increased, but nothing of the kind has occurred." Eber says: "After all, there can be no doubt that neither the subcutaneous injection nor the effect which this agent may induce in a healthy subject or in animals affected with tuberculosis, with the sole exception of the extremely tuberculous (cachectic), carries with it any risk that can prohibit the extensive use of tuberculin in the crusade against tuberculosis in cattle." Nocard, also, in 3,000 cases has seen but three instances of injury to health, and these were all cases of extraordinarily advanced disease. But in man, also, tuberculin as a diagnostic agent has been extensively used, notably in Koch's Institute, and at Koch's Institute no single instance of any injurious effect or generalisation has been observed in the thousands of cases so treated, nor is there any record of any case in which the hypothetical transference of bacilli to distant organs has taken place with the result of fresh lesions appearing at a distance from the primary seat. Koch has himself expressed the opinion that there is no evidence to support the far-fetched hypothesis that tuberculin causes mobilisation of the tubercle bacilli; and indeed, although tuberculin has been used by many, we never hear nowadays

of this risk. I have used tuberculin hundreds of times without any such result. One patient of mine used tuberculin on and off for six years. His lungs were riddled with cavities, and he maintained that nothing but tuberculin did him any good. In spite of his advanced disease, the tuberculin always caused improvement, and there was never any sign of mobilisation. On the contrary, I am convinced that tuberculin prevents rather than causes the mobilisation of tubercle bacilli. We may conclude, then, that tuberculin as a diagnostic agent is safe and sure in the hands of those qualified to use it.

So far as my own opinion and practice are concerned, I may add that the use of Old Tuberculin as a diagnostic agent goes hand in hand with the treatment of tuberculosis by New Tuberculin. We who believe in the specific treatment of tuberculosis use every means to detect tuberculosis in the latent form, before it causes any definite symptoms, and then it is that we claim effects that cannot be produced by any other known method of treatment. It is in such cases that the triumphs of Koch's great discovery will be demonstrated, as soon as observations are established on thoroughly logical lines. In a scientific sense we can only speak of the cure of tuberculosis if the reaction has been obtained at the outset of treatment, but fails to show itself after treatment. The tuberculin test, therefore, is the criterion also of successful treatment. It is known that many cures by climate, sanatoria, etc., would not stand this rigid test, while many cures by tuberculin have already satisfied this test. But I do not presume to say that the chapter on the treatment of tuberculosis begins with specific diagnosis and ends with specific treatment. A large chapter remains to be written, both on the methods of common sense and on the methods of science; but the chapter on the value of Old and New Tuberculin in diagnosis and treatment is well worthy of consideration. Tuberculosis of the lungs is such a variable condition, and runs such a variable course, sometimes over a space of months, sometimes over a space of years, that it must be many years before any just judgment can be formed on the value of any method. There are many who have condemned

tuberculin without a trial. There are some who have treated tuberculin to a trial on a par with the scandalous trial of Dreyfus in France. Even though Dreyfus may never find justice at the hands of France, tuberculin will outlive those who despise and decry it, and will prove itself to be an invaluable agent, both in diagnosis and even in treatment.

(2) Next, is tuberculosis so common that the tuberculin test loses its value altogether as a diagnostic agent? This argument is much more solid and more difficult to dispose of, especially when we bear in mind that even a tuberculous lesion in man may be due to the bovine type of the tubercle bacillus and cause a reaction to tuberculin, although such a lesion has little or no tendency to progress in adult human beings. Now, if we accept the view, as I do conscientiously, that at least pulmonary tuberculosis is rarely related to the bovine type of tubercle bacillus, and pulmonary tuberculosis represents eleven-twelfths of all the tuberculosis that occurs in man, we at once reduce this error to one in twelve. Next, as far as observations in adults go, the great majority of tuberculous lesions outside of the lungs are also due to the human type of bacillus. Thus the margin of error is still further reduced, though as yet we cannot fix accurately the degree of this reduction. So far as statistics help us, the bovine type does not appear in more than one-third of the lesions, other than those of pulmonary tuberculosis, even when the statistics have been drawn from cases in which special pains were taken to discover lesions that were more likely than others to yield the bacillus of the bovine type; and these cases were almost wholly children, not adults. Both the investigations of the German (Kossel and Weber) and English Commissions, little as they may agree on other points, agree on this point, that even in young children the human type is more commonly found than the bovine type. Accordingly, we may admit that once in 30 cases or thereabouts the tuberculin reaction may be due to the presence of bovine tubercle bacilli, when children are included. So far as pulmonary tuberculosis alone is concerned the risk of error due to bovine bacilli may be almost ignored. Spengler's views that both types are to be found in sputum has not been confirmed. Of course

if we imagine that there is no essential difference between these two types—human and bovine—and that one can be readily converted into the other type by the mere change of soil, this margin of error vanishes—in other words, tuberculin is a certain test of the existence of a tuberculous lesion whether due to tubercle bacilli of the human or bovine type. We may thus further simplify the solution of our problem and discuss the question whether latent tuberculous lesions are so common in apparently healthy adults that the tuberculin test is altogether too sensitive a test to be of any use in diagnosis.

In the first place, we do not propose to use tuberculin indiscriminately in the community. If we did we could do no harm, for the healthy would not react, and we should know accurately to what extent tuberculous lesions of some sort or size were present in the apparently healthy. I have tested myself already, and after a sufficient lapse of time I shall test myself again, for as soon as ever I find that I react, I shall give myself a course of tuberculin treatment. I can see no virtue in a lesion that harbours living tubercle bacilli, and whenever I prove the existence of such a lesion in my body I shall proceed to help nature to get rid of the offensive material. Of course I hold that the specific reaction only occurs when there are living tubercle bacilli somewhere in the body.

Who can tell whether the lesion in this body or in that body will remain latent or become obsolete? The dark records of statistics tell us in black columns that this very disease is responsible for about one-seventh of the whole mortality in civilised communities, and, worse feature still, most of its victims are done to death in the very prime of life. The conscientious physician should be over-zealous in his efforts to rescue this enormous contingent. Is he to relax his efforts at saving life because, forsooth, in his efforts to save life he may offer a helping hand even to one who needs no assistance? It is surely sound practice to treat every member of a family of ten in order to save the tenth, when one is satisfied that the treatment in itself can do no harm. Let me appeal to the over-zealous surgeon for his opinion. Nowadays almost every tenth person one meets

has been operated upon for appendicitis. In simple appendicitis the mortality is hardly 1 in 10, if no operation be done. The over-zealous surgeon thinks that every case of simple appendicitis should be submitted to the operation for removal of the appendix. This means that 90 out of every 100 operations are unnecessary. The operation itself is by no means free from risk, and yet ten operations on ten individuals are necessary to save one life—and occasionally it will happen that an individual will lose his life because the operation has been done. Now whatever line of argument be used, there is no suggestion as yet that 97 per cent. of every community should be tested with and treated by tuberculin. I use the figures 97 per cent. because everyone, without even taking the trouble to learn for themselves what Naegeli found and what he inferred, talks glibly of Naegeli's astounding figures of 97 per cent. of tuberculous lesions in the few hundred hospital cases that were examined by him in the mortuary at Zurich.

The statistics drawn from a hospital in a very poor district are no guide to the relative prevalence of diseases in a large community. Naegeli's figures were drawn from the dead and not from the living—from the very poor, who suffer far more from tuberculosis than the well-to-do or rich—and from lesions which were not in every case proved to be caused by tubercle bacilli. When, therefore, we suggest tuberculin for diagnosis, we do not expect to use it in any wholesale way, nor is there any urgent reason for using tuberculin as a diagnostic agent until the patient comes to the physician for advice. When the patient comes, he is probably not in good health, nor does he understand why his health has suffered. Then it is that the wary physician cannot ignore the common disease, tuberculosis. It is no rare experience for the physician to find truly advanced pulmonary tuberculosis when the patient has never before consulted a physician, but often the disease is not advanced and the symptoms may be a cough, or loss of energy, loss of appetite, indigestion, shortness of breath, pain in the chest, rheumatism, palpitation. There may be little or no cough or phlegm, and then suddenly an acute pleurisy, or a severe hæmorrhage or a pneumo-thorax

may be the accident that reveals the serious condition of the lungs. In these insidious forms of pulmonary tuberculosis there is a stage of latency when the lung is hardly damaged which nothing but tuberculin can detect. Is it of no advantage to detect the disease before the lung has been seriously damaged—especially if one is convinced, as I am, that tuberculin used as a remedy may effectually prevent the progress of the disease at least for many years? Does it matter very much if every now and then a case is treated that would not have progressed at all? It is certain that no one can tell in which case the disease will progress or in which it will become latent or die out. Spontaneous cures do occur in the very early stages in perhaps 25 per cent., but this is tantamount to saying that 75 per cent. fail to heal spontaneously. How often does the pathologist find that the disease has been arrested in the second or later stages?

Let us even allow that 50 per cent. get well by Nature's simple process, 50 per cent. progress and ultimately cause death. Surely it is worth our while to use tuberculin for diagnosis and treatment, if by treating a hundred cases we can make sure of checking the disease in most of them. If we do not use tuberculin for diagnosis or treatment there is no other way of curing all cases in the early stage. In this respect sanatorium methods fail, as Krause, Rembold, Weicker, Moeller, and others found. I admit that before these important admissions will be made it is necessary to be converted to the view that tuberculin, both in diagnosis and treatment, does nothing but good. Such a conversion must be a tedious process for most medical men, who have the temper of saints, perhaps, but the temper of St. Thomas Didymus rather than of St. Paul. On this point my mind is quite clear, that until tuberculin is used steadily for a year or more the startling truth of its value, both in diagnosis and treatment, cannot be realised. I would lay any ordinary wager that no one who has had the courage and patience to use tuberculin, or to watch its use for a year, would have any doubt of its great virtues. Even in those very early cases, when the lesion may be quiescent, and the symptoms and physical signs not too definite, no harm can

come from the use of tuberculin in diagnosis, and enormous good may come, because the disease can be arrested before the lungs have been seriously injured. If we can raise the percentage of permanent arrests of the disease by 50 per cent., it is well worth doing this at the risk of treating some cases that would have come to a standstill without the artificial use of tuberculin at all. We have already said that ninety unnecessary operations for appendicitis are performed in order to save at most ten lives, and this operation is not free from risks even to life. Surely, then, we may urge the use of tuberculin if we find that it saves far more lives without any risk at all.

But after all, the value of tuberculin in diagnosis is not limited to the minority of cases, in which there are neither definite symptoms nor signs. Unfortunately not one case in ten belongs to this category. Its value is often great when there are definite symptoms and signs. I have thus been able to exclude tuberculosis and pronounce in favour of hydatid disease of the lungs—especially when there is constantly recurring hæmorrhage. In bronchitis—even localised bronchitis due to influenza bacilli, pneumococci and streptococci—tuberculin may be used for diagnosis when the fever has passed away. Pulmonary tuberculosis or bronchial tuberculosis in children may simulate asthma, and tuberculin may rapidly relieve the asthma. But both in adults and in children localised caseous deposits—small enough to escape detection, and perhaps causing no obvious signs or symptoms—may suddenly break down and burst into the blood-vessels, causing generalisation, or into the air passages, causing acute tuberculous bronchopneumonia. Such lesions early recognised and treated by tuberculin may be rendered harmless. In my experience I have had no instance in which doses of tuberculin have been followed by any symptoms that would suggest that the tuberculin had thus set free masses of tubercle bacilli. No doubt such symptoms may have occurred after doses of tuberculin, but why should this effect be attributed to the tuberculin when it is such a common accident? I repeat, that in my opinion tuberculin in proper doses tends to prevent such an accident, as it prevents hæmorrhage and tuberculous menin-

gitis by favouring the fibrous organisation of a tuberculous formation.

Tuberculin is invaluable in the early stages of pulmonary tuberculosis when the symptoms are related to other organs—heart and blood-vessels and blood, digestive organs, especially the stomach, and even the muscles, nerves, bones and joints—especially symptoms of the rheumatic type. The enormous improvement in the general health—gain in weight and especially gain in energy—and the complete disappearance of the rheumatic pains must profoundly impress anyone who has witnessed the remarkable change from invalidism to first-rate health.

By using tuberculin we may correct the wrong diagnosis of those who trust to the superficial evidence of physical signs. I have thus saved many a person not only the worry and anxiety "inseparable from the consciousness of having consumption," but also the expense and trouble of a visit to the country. It is past my comprehension why tuberculin is not freely used in every general and special hospital, and above all in special hospitals for pulmonary tuberculosis and in children's hospitals. But while I differ from Nägeli's inference that "jedermann ist ein bisschen tuberculös," latent tuberculosis is common enough, and may at any time become active. Professor Hansemann relates a case in which tuberculous meningitis supervened in a child soon after a serious fall upon its head. At the post-mortem examination the *foci of virgæ* of the tuberculosis was found in a caseous bronchial gland at the hilus of the lung. It is a matter of everyday knowledge that an acute inflammation of the lungs, due to streptococci, pneumococci, influenza bacilli and whooping-cough may convert a latent into an active tuberculosis of the lungs. Lydia Rabinowitch tells us that living tubercle bacilli may be found in the hard calcified masses of tuberculous lesion, and more recently Lubarsch has shown that both in man and bovines the tubercle bacilli may retain both their vitality and virulence in these hard masses. Until the true import of these latent foci, which harbour living and virulent tubercle bacilli, as frequent sources of active and even acute pulmonary tuberculosis be recognised, the full value

of tuberculin in early diagnosis and treatment cannot be appreciated.

The enemy may lie in ambush—even in a dense bed of chalk—for many years, but is nevertheless a source of constant danger. A wise man will design to destroy the enemy before it has the chance of doing mischief. The only weapon for such a purpose is tuberculin. In this way also tuberculin becomes an instrument of pre-eminent value in prophylaxis. "Every latent focus deprived of its living bacilli" is one potential source less of pulmonary tuberculosis. Thus the early treatment of tuberculosis by means of tuberculin is a real step forward in prophylaxis—of far greater efficacy than sanatorium methods in preventing the escape of tubercle bacilli at a later date from early lesions and also in converting the open into the closed form of pulmonary tuberculosis.

APPENDIX OF ILLUSTRATIONS

Instances in which pulmonary and other forms of tuberculosis have not been recognised as such, until tuberculin was used for diagnosis.

Healthy 1. 1903. N. (Stage I., Case 11), rejected for sanatorium as healthy.
Lungs. Evident physical signs and enlarged cervical glands. No Th. at first.

Tested: 1000cc. 105°.

Once afterwards Th. found.

Treated with tuberculin; gained a stone.

Healthy 2. 1907. Miss O. R. Dr. E. and Dr. M. said lungs
Lungs. were healthy. Signs of R. L. No Th.
 1000cc. Old T. 99.4°.
 1000cc. 101°.

Treated with tuberculin; very sensitive.

Gastric 3. 1907. A. W. (Stage II., Case 80), 22. 6 st. 13.
Ulcer. Diagnosed as "gastric ulcer" by two doctors. Operated on for appendicitis by one doctor, but no better.

R. 1-11. Enlarged and painful cervical gland. Symptoms of severe nervous dyspepsia (pain, vomiting) greatly relieved by lavage of stomach. Also henteric diarrhoea. Menies irregular. From 13-17 years of age quite regular; not unwell from 17-18. Since 18 (five years), one every five months. In October, 1907, after tuberculin treatment, menses became regular again and remained so till present (March, 1909). Now also (March, 1909) cervical gland, which was as large as almond, cannot be felt.

Pain in abdomen so severe that a surgeon operated on her for appendicitis. "Just as bad after operation."

Surely better to give a test dose than operate for imaginary appendicitis.

Tested: May, 1907. Weight, 6 st. 12.

1000cc. Old T. N.

1000cc. 100.2°. Arms much inflamed.

Treated up to 1250cc. P.T. Gained 16 lb.

March, 1909. Weighs 8 st. 11. Has lost gastric symptoms.

Am testing her again.

4. 1904. J. E. K., 16.

Hemorrhage. Consulted Dr. R. for hemorrhage. Dr. R. found from throat, nothing in lungs to account for hemorrhage. Said "blood came from throat," and sent patient to specialist (Dr. S. K.), who also said blood came from throat. Not satisfied, came at once to me.

I found half a dozen tubercle bacilli in bloody sputum.

5. 1911. Mrs. B. Diagnosed as pleurisy with effusion,

Pleurisy. though medical man did not suspect tuberculosis. Weight 7 st. 15.

Made no progress in hospital till I tested her and treated her with tuberculin. No tapping; yet fluid entirely absorbed, and she gained 30 lb., pregnancy accounting for some of this increase.

6. 1902. Miss W., 26. Weight, 7 st. 12½ (Stage I.,

Acute Case 6).

Rheumatism. Brother shortly after died of tuberculous disease of spine. Diagnosed by three doctors as acute rheumatism. Pulse, 120. Temperature, 99.5°, 100°.

Tested when temperature fell to normal, because I found signs of L. I-II.

000cc. T.A. 99.8°. 000cc. T.A. 100.2°.

005cc. " 100.4°. 001cc. " 102°.

01cc. " (repeated) 103.4°.

Treated with tuberculin. Lost all rheumatic pains; gained 26 lb. Weight, 9 st. 10½, and was well in 1908.

7. K., 26. 8 st. 4.

Pneumonia and Pleurisy. Diagnosed as "pneumonia and pleurisy," after temperature subsided.

Tested: 000cc. Old T. 100.8°.

Severe general reaction.

Treated with tuberculin. Lost cough; recovered strength; and gained 28 lb.

8. May, 1902. E. R., 3. Weight 5 st. 5.

Asthma. One doctor diagnosed "asthma," one "bronchitic." I found signs R. L-I-I. and bronchial tuberculosis.

Tested: 000cc. Old T. 98.4°. 001cc. 99°.

000cc. " 103°.

Treated with tuberculin. Gained 14 lb.; 4 st. 5½.

Tested in October, 1907. No reaction to 01cc. Old T. Weight, 5 st. 13. March, 1909, well.

9. 1902. F. P., 14. 4 st. 2½.

Typhoid Fever. Admitted to hospital as "typhoid fever." Found signs of pleurisy. After fall of temperature, tested. Reacted.

Treated with tuberculin. In nine months gained 40 lb.

Weight, 7 st. Tested a year later. Reaction doubtful.

10. 1904. Miss B., 22. 8 ft. 6.

Diagnosed by first doctor as impacted calculus, and sent to hospital for operation. Signs found by me later.

R. 2-14, L. 1, and extensive pleurisy at base.

Treated. Several reactions. Gained 28 lb. Got married, and has a child. Well in 1909, but should be tested.

11. 1904. M. C., 28. 7 ft. 12.

Sent to hospital by Dr. L. for "gastric ulcer"; aching pain in epigastrium. Good appetite, but vomiting all food.

Tested: voice. Old T. 953'; voice. 1002'.

Treated with tuberculin. No treatment of stomach. In four months after tuberculin gained 27 lb., and gastric symptoms disappeared. Weight, 9 ft. 11½.

12. 1904. N., 22. 5 ft. 9.

Sent to hospital for gastric ulcer. Pain in stomach after food, and vomiting.

Tested: voice. Old T.

Treated with tuberculin. Gained 20 lb.; lost cough and gastric troubles. "Feels quite well"; was well in 1907.

13. 1904. K. S., 20. 6 ft. 11.

Anæmia. Anæmia for two years. R. 2,800/500. W. 10, 520. Hb. 35%; colour index .6.

Tested: voice. Old T. 1002'.

Had Blood's Pills and T. E. Est. (T. E.) 10-0400. Gained 10½ lb.

14. 1905. J. P., 21. 4 ft. 8.

Pneumonia. Saw in consultation as case of "pneumonia." Seven other doctors said "pneumonia." I at once suspected tuberculosis. After temperature normal—slow descent—tested.

voice. Old T. 103'.

Treated with tuberculin. Within a year gained 40 lb. Now alive and well.

15. 1905. C., 31.

Peripheral Neuritis. Diagnosed as "peripheral neuritis" by neurologist. I said certainly also "myelitis," but found latent pulmonary tuberculosis (L. 113) and therefore diagnosis tuberculosis-meningitis.

16. 1906. W., 26. 11 ft. 12.

Treated as gonarthral rheumatism for 10 months. Then I saw in consultation.

Tested: voice. Old T. 1004'.

100500. " " 998'.

Quite against Wright's ideas.

voice. 103'.

Also a perineal sinus leading prostatic abscess. Treated with tuberculin. In five months gained 25 lb. and joint greatly better, and perineal sinus closed.

17. 1907. M., 40. 6st. 7.

Copious chronic pleural effusion. Three pints drawn off.

Tested: 100cc. Old T. 98.4°; 100cc. 98.4°.

100cc. 100.6°.

Treated with P.T. up to 55cc. Within six months, weight 8 st. 6½, and very great improvement. Gained 27 lb.

18. Mrs. D.

Pleurisy. Treated by six doctors for pleurisy. Now in Stage III, pulmonary phthisis.

19. E. D., 41. 9 st.

Diagnosed as Cerebral Tumour. Headache, frontal; vomiting of cerebral type, without warning or nausea, and unrelated to food; not dull mentally; no fits; slight goiter, pupils normal. No optic neuritis—reflexes normal. Temperature and pulse normal.

Tested: 100.5cc. Old T. 99.8°. Local reaction in arm.

100.5cc. " " 101°.

In nine days tuberculin used, then gained 7 lb. in three weeks. Symptoms disappeared. Gave up coming.

20. 1906. L. T., 27. 5 st. 6½.

Pain in stomach, vomiting, flatulence, dirty tongue, constipation, diarrhoea, abdominal pain. T. 99.4°-100.2°.

E.v. 57. N. 100.5. Old T. 100.8°. 6 st. 6½.

13 " N. 100.5cc. " N.

17 " 99 " 100.5cc. " 99.8°. 6 st. 6½.

20 " N. 100.5cc. " 100.4°. 6 st. 5½.

27 " N. 100.7cc. " 100.2°. 6 st. 5½.

Then P.T.O. in increasing doses without any reactions till

2.14.07. N. 35cc. P.T.O. 100.2°. 7 st. 1½.

3.14.07. Feels better, but still some pain in abdomen.

Went out of hospital.

21. 1907. F. 19. 8 st.

Acute Lead Poisoning. Acute lead poisoning. Tested after convalescence because enlarged glands in cervical triangle and groin.

100cc. Old T. N. 8 st. 2.

100cc. " 101.4°. 8 st. 2.

Treated with P.T.O. In two months 8 st. 10½, and feels very well.

22. 1902. B., 29.

Dr. C. removed large cervical gland.

Subsequently signs in both apices and reaction to tuberculin.

Strained voice.

23. 1902. Mrs. L., 38. 9 st. 10. "Strained voice."

Found paralysis of right vocal cord. Also signs of pulmonary tuberculosis at right apex (R. 1-11). Re-

current laryngeal nerve no doubt involved in fibrous formation.

Reacted to :000cc. Old T., 105'.

Treated with T.R. Further reactions. Great improvement, and well for more than six years, though voice still cracked. 1909—very well.

24. 1901. C.

Asthma. Had also suppurative disease of nasal sinuses and polypi.

Tested : :000cc. Old T., 105'.

Severe reactions with herpes.

Treated. Remained well for year; also removed polypi, and deak radically with sinuses.

Tested in 1905. :01cc. Old T., 102'. Treated again 1909. At work as insurguard.

25. W. C., (November, 1905), 45. 3 st. 15.

Very obscure case. Hot feeling in right shoulder, right foot sore, aching pain in feet; both knees. Localised tenderness over spine, and localised pain shooting out on one side in region of dorsal vertebrae.

Tested : Reacted to tuberculin (100').

Treated with tuberculin. Pains abated, and other symptoms improved; gained weight and improved, but left off treatment.

26. 1905. Mr. C., 21. 12 st. 5.

Healthy lungs. Four doctors pronounced lungs healthy.

Tested: :000cc. Old T., 102'2". Very severe reactions and swelling in arm, yet passed his own doctor, also railway medical officer, Dr. B. and Dr. D.

Treated with tuberculin. Several reactions. 100', 102', 105'. Yet two years later no cough. Recovered all energy, and feels perfectly well.

27. 1905. Miss C., 17. 3 st. 11.

Influenza. Diagnosed as "influenza."

Tested: :000cc. Old T. 99'5".

:000cc. " " 99'5".

:005cc. " " 104'.

Had been treated for ordinary cough and cold for three months without any benefit. Improvement began immediately after use of tuberculin: gained weight, lost cough, and perfectly well three years later. 1909—perfectly well.

28. 1906. Mr. H. (March, 1905), 20. 9 st. 12.

Tested: :000cc. Old T. 102'.

Treated for some months. Felt quite well.

29. 1904. A. W. (September, 1905), 18. 8 st. 14.

Anæmia and Debility. Tested: :000cc. Old T. 101'.

Treated with tuberculin. Recovered health and strength. 1909—quite well.

30. S. H., 12.

Pleurisy with effusion. Pleurisy with effusion. Temperature, 99.4°-100°. Tested: (Temperature was N. for few days).

100jcc. Old T. 99.2°.
 1000cc. " " 99.8°.
 1002cc. " " 99.4°.
 1005cc. " " 100.4°.

31. 1902. C., 38.

Cirrhosis of Liver. Admitted for cirrhosis of liver, and showed symptoms of such. Temperature, 100°. History of jaundice. I found abdomen full and tender in flanks, and felt a distinct mass above and to right of umbilicus.

Tested: 100075cc. Old T. 100°. Next day 100°, 100°; third day 98°. Marked improvement under tuberculin treatment, but gave up treatment because he felt able to work.

More than two years after first coming to me, was advised by another physician to submit to laparotomy, and died the day after the operation.

32. E. W., 6, 23.

Bronchial tuberculosis and hydatid of lung.

Tested: Reacted.

Treated with P.T.O. and then P.T.R. Em. Gained 10 lb.

Operation for hydatid at right apex. Now quite well.

33. Miss D. G., 20, 5 st. 7.

Doctors could find no evidence. Physical signs at right apex (R. L.-II.). Tested: 100cc. Old T. 101°.

Treated. Sensitive to T.R., especially subjective disturbances, pains. Lost all symptoms. Well three years later.

34. 1906. Miss D., 26, 5 st. 5.

Enlarged cervical glands removed by Dr. Mac C. in August, 1905. In May, 1906, came to me. Found other glands on same side enlarged—under sternomastoid, over scapula, under clavic. Found physical signs (R. L.-II.). Drs. Mac C., L., and D. found nothing in lung.

Tested: 100cc. Old T. 101°.

Treated with T.R., P.T.O., and P.T. Great improvement.

After foolish walk up high mountain, lost ground, and one large gland softened, and was removed by Dr. M.

Two years and nine months later all glands have become small, and patient looks and feels well. 1909. Looks well, but further treatment was given.

35. 1906. F. G., 10 st. 8½.

Two doctors examined lungs and found nothing. I found definite physical signs (R. L.-II.), which doctors subsequently admitted.

Tested: 100cc. Old T. 101°.

Arm very much swollen.

Treated with P.T. Lost severe cough and phlegm, and improved in health. 1909. Apparently well; did not react to '05cc. Old T.

26. 1906. Miss B., 19. 7 st. 1½.

Rheumatism. Hip disease at 16. Pulmonary tuberculosis. Stage II. Treated for rheumatism for three years.

27. 1907. Mr. H., 38. Post 1.

Pt. got a first-class life policy in A.M.P. Society the day before he saw me, and Dr. H. said "there was nothing the matter with him." Found signs of early disease (R. I-II). Tested. '000cc. Old T., '000'4. When I gave my opinion, patient was sceptical, because one doctor had sworn his lungs were healthy, and the other said he was suffering from catarrh. In order to prove that I was right, I gave him '003cc. Old T., '003'4. A very severe reaction—fever, vomiting, severe headache. All scepticism set at naught, and specific treatment took away cough, and he gained 6½ lb. in a month.

29. 1907. R. W., 17. DIAGNOSIS as Gonorrhea.

Tested '00075cc. Old T. 98'4.

'003cc. " " 108'8.

'00200 " " 98'4.

'00400 " " 100'4.

Came into hospital a year later, when she still reacted.

30. 1905. Miss S., 28. 7 st. 6. (Case No. 14, Stage III.) Obscure history. Was operated upon for floating kidney, but operation for fusion did no good.

Saw Dr. S., who suspected hydatid disease of lungs. Sent into Prince Alfred Hospital. Needles were inserted into base of right lung several times; nothing found. Went out, and came to me. I found signs of pleurisy at right base, and very obvious signs of a cavity in left lung, extending down to third rib and outwards into axilla. Might be a bronchiectasis or abscess cavity after rupture of hydatid cyst. There had been history of coughing up suddenly fluid, and then "skins." Sputum was very abundant—about a pint daily of "anchovy sauce" pus, containing beautiful chains of streptococci, growing readily on media, (not streptothrix). No signs of hydatid. Tubercle bacilli could not be found, in spite of numerous examinations. Later, tubercle bacilli found *em.* I did not see the specimen. Well marked clubbing of finger-tips.

Tested. '000cc. Old T. '05'.

Treated with P.T.O. Very sensitive.

Temperature 103°, 102°, 100°, but nevertheless gained 24 lb., and in course of months sputum reduced in amount from more than a pint to half an ounce. Weight 1 st. 12. Then treated with P.T. up to 2 cc. She said she "felt so strong after last big dose."

Tuberculosis was not thought of until I tested her, and specific treatment did wonders for her.

41. 1905. Mrs. M., 39. 6 st. 5.

Debility. Treated for "debility." Physical signs at right apex (Stage I). No Tb.

Tested: voccc. Old T. 100°4'.

Treated with P.T.; then T.R. Em. up to 4 ccm.

In five months, weight 6 st. 12½. "Can walk splendidly now; could hardly walk at all, when she came first." In six months, weight 7 st. 7½. Gained 11½ lb., and feels quite strong.

42. 1907. Miss W. D. (sister of above), 19. 5 st. 6½.

No sign of lung disease. Saw Dr. Angel Money, who found no evidence of pulmonary disease.

Tested: voccc. Old T. 100°2'.

voccc. " " 100°.

voccc. " " 102°.

Treated with T.R. and T.R. Em. Gained 5½ lb. Quite well six years later. 1909—quite well.

43. 1903. Miss K. (October, 1902), 30.

No lung disease. Treated by Dr. M., who said there was "no lung disease."

Tested: voccc. Old T. 102°6'.

Treated with T.R., T.R. Em. Gained weight and recovered health.

44. 1904. S., 17. 8 st. 12½.

Enl. cervical gland and pulmonary tuberculosis (R.L.). Tested: Reacted severely. voccc. Old T. 101°. Very great improvement under tuberculin treatment.

45. 1904. E., 24. 9 st. 6.

Pulmonary tuberculosis. R. I-II. No Tb. voccc. Old T. 99°8'. Treated with T.R. emulsion, sensitive, then P.T. 50-175 ccm.; reactions 1909—well.

46. 1904. Miss B., 49. 7 st. 3½.

Dyspepsia and Bilious attacks. Tested: voccc. Old T. 100°; voccc. T.R. Em. 102°.

Treated: Lost all symptoms, and gained 10½ lb.

47. 1907. Mrs. C., 22. 8 st. 1.

Weak Lungs. Dr. P. and Dr. McC. (surgeon). Weak lungs.

Tested: voccc. Old T. 98°4'.

voccc. " " 100°.

voccc. " " 100°.

voccc. " " 101°.

Treated: Improved and gained 7 lb. 1909—well.

48. 1906. J. E., 21. 7 st. 15. Diagnosis: gonorrheal rh. by several surgeons.

Tuberculosis of wrist joint. A scar in palm of hand near thenar space left by a healed sinus.

Left wrist swollen and tender. Much impairment of movement.

Tested: 100cc. Old T. 102°.

100cc. " " 103½°.

Wrist greatly improved under treatment, and gained several pounds in weight.

42. 1905. Miss R., 26. 7 st. 6.

Pulmonary Treated for some time for asthma by Dr. —.

Tuberculosis Tested: 100cc. 98°.

diagnosed 100cc. 100½°.

as Asthma. Also 100cc. T.R. 101½°.

Treatment very successful. Asthma ceased, and five years later weighed 9 st.

50. 1906. Miss D., 27. 8 st. 6.

Indigestion. Lost weight, and symptoms of "indigestion."

100cc. (Old T. 100½°).

Treated with tuberculin. Before treatment was losing weight at rate of a pound a week for several weeks. Gained weight steadily under tuberculin treatment. Suffered much from rheumatic pains, which disappeared under tuberculin treatment.

51. 1907. Mr. P., 33. 10 st. 2. (N. 11 st. 7.)

Pulmonary Sister 15 years ago.

Tuberculosis, Examined by railway medical officer, who said that he called was well able to work. Cough for six months. Pleurisy malingering. three months ago. "Great pain;" "could feel grating inside."

Tested. Reacted.

Treated with tuberculin. P.T.O. up to 1 ccm.; gained 15 lb.; then P.T. up to 1 ccm. Weight, 10 st. 10½.

"Feels splendid now; eats and sleeps well."

52. J.H., 21. 7 st. 11.

Rheumatism Treated by Doctor C. for rheumatism for five months, then sent to hospital as "typhoid fever." Temp. 102°.

Fever. Signs of pleurisy and also of pulmonary tuberculosis at both apices. No Tb. at time, though later, when temperature became normal.

Tested: 100cc. Old T. 101°.

Treated with tuberculin. Many severe reactions; but in a year weighed 9 st. 5; gained 22 lb. Enormous improvement in every way.

53. Mr. C., 25. 9 st. (July, 1907).

Catarh. Physical signs at right apex (R. L-11.).

No Tb.

Tested: 100cc. Old T. 99°.

100cc. " " 98°.

100cc. " " 98½°, 101°, 105°.

Next day: 101½°, 106½°.

Cough worse.

Treated with success.

54. Miss C., 22.

Anæmia, perhaps Pernicious Anæmia. Sent by doctor for "anæmia," perhaps "pernicious anæmia." Physical signs of R. II. Great pallor. Temp. 98° to 100°. Treated with P.T.O. Gained 13 lb. in six weeks. Treated with P.T. Lost all symptoms, and feels as well as ever in life.

55. Miss R., 32. 12.

Disease not suspected till test.

Tested: Reacted to 1037°. Herpes, and very severe symptoms.

Treated with T. R. Dose up to 35ccm. Gained 17 lb. Well.

55a. Mr. D., 25. 94. 105.

Physical signs negative. Did not think he had any disease. His sister was under my care, and knowing the benefit she had received, he requested me to test him—to be certain.

Tested: Reacted to 100cc. Old T. Felt very ill: headache, and severe reaction in arm.

Treated: Gained weight, and felt splendid.

56. Mr. R. (August 1907), 22. 12. 60. 1.

Doubtful signs (R. I.). Symptoms of low fever two years before; night sweats and evening rise of temperature. Short of breath. Lost 19 lb. No Tb.

Tested: 100cc. 99°. Next day 97° 2', 99° 2', 99° 4', 100° 4', 100° 4', 99° 8', 99° 4', 99° 8', 99° 5', 99° 8', 98° 8', 98° 8'.

Treated. Very great improvement.

57. Miss B., 22. Sister.

Physical signs (R. I.-II.). No Tb.

Tested: 100cc. Old T. 100° 4'.

Treated. Extremely sensitive. While in hospital under treatment caught typhoid fever from patient, and had a very severe attack, lasting three months. A year later saw her. She had put on much weight, but still, although very heavy and not feeling ill, reacted very severely to 100cc. Old T. (1037°). Since treated with P.T.O. and P.T., with great improvement.

58. Miss W., 22.

Mother in Stage III.

Tested: 100cc. Old T. 104°.

59. Miss M., 20. 1906.

Mother do; and five years later, her husband, who "caught it from her."

Physical signs: R. I.-II.; ribs.

Tested: 1906. Old T. 98°4.
 1907. 1917. Cough worse.
 Treated with success.

60. L., 43.

Called Enlarged glands in neck and axilla of right side. I
Lymphadenoma also found well-marked displacement of heart due to
 pleural effusion. Friction also heard at right base.

Pt. had then seen four doctors. One, Dr. T., a
 gynecologist, diagnosed "indigestion" without discovering enlarged
 glands in neck. Drs. H., M., S., and F. had diagnosed "lymphadenoma"
 without examining blood.

When he came to me I tested him there and then.

1906. Old T. 100°8', 101°, 102°, 97°, 98°4', 98°4'.

I told him he had tuberculous pneumonia, and probably glands were
 also tuberculous. Pt. thinking truth must be with majority, consulted
 another doctor—Dr. F.—who scouted the idea of pleural effusion, in spite
 of friction sounds, dullness and displacement of heart. Pt. Therefore
 returned to his former doctor—Dr. H. M.—who treated him for
 lymphadenoma. Within two months Dr. H. M. himself found fluid in
 pleura, and advised tapping. This discovery again of fluid, which I had
 discovered two months earlier, disconcerted patient, and he went to
 another doctor, who drew off fluid. When he was well enough, the
 patient came back to me, and repeated his experience in the presence of
 another doctor—Dr. G.—certainly not an experience likely to impress a
 layman with a high opinion of the competence of medical men to pro-
 nounce upon physical signs. Dr. then examined blood, and excluded
 "lymphadenoma."

Here again the tuberculin test settled the point upon which there had
 been such a remarkable difference.

61. Mr. S., 33. 3rd 12.

Inflammation Inflammation of lungs in March, 1908. Six weeks
of Lungs. Ill; never lost cough; much blood in sputum. Seen
 away. Came to me.

Tested: 1906. T.O.A. 98°4.

1906. T.O.A. 98°4.

1907. T.O.A. 101°.

Treated with tuberculin. Lost cough, and recovered health.

62. Miss A., 26. 7th 3.

Sciatica. Treated for severe sciatica. Signs: R. II, L. II, 0.
 Fleeting, pain, great wasting, clankers, cystitis.

Tested: Reacted severely.

Treated for some time with tuberculin. Improved, and then relapsed.
 Then refused further treatment.

63. Miss W., 26. 6th 10.

One brother 49 in right month. Father and father's brother 46.

Physical signs (R. L.-H.): Evening rise of temperature. Pulse, 120-125.
 Tested: 100cc. T.A. 996°.

10025cc. 10014°

Dr. S. said, "Mischief in lungs."

Dr. B. said, "Had been mischief, but was arrested."

Dr. P. said, "No mischief."

Treated with tuberculin. Several reactions (1102°, 1006°, 101°), yet gained weight. Wt. (VI. 08). 7 st. 12.

Mother says she looks and feels a great deal stronger.

64. Mrs. C., 44. 7 st. 6.

Physical signs (R. L.-H.): c. riles.

Treated for indigestion for long time.

Tested: 100cc. Old T. 100°.

1001cc. " 101°.

1003cc. " 102°. Cough worse.

Treated 1909. No cough—no indigestion—feels well.

65. Miss McC. 5 st. 5.

Treated for asthma. Loss of strength and of colour; breathless.

Tested: 100cc. Old T. 9980°. Very severe reaction in arm.

1003cc. P.T.O. 1004°

Treated with P.T.O. and P.T. up to 5cc. Recovered health, and weight 9 st. 4.

Tested, February, 1909. Negative to 105cc. Old T.

These records show that in the earlier stages of pulmonary tuberculosis and of other forms of tuberculosis mistakes are inevitable and not uncommon unless tuberculin is used as a diagnostic agent. In all my experience with tuberculin as an aid in diagnosis—and I have used it in many other cases than those recorded—I can state positively that the mere use of tuberculin for such a purpose is quite free from danger of any kind provided it is used according to conditions and limitations imposed by Koch himself twenty years ago. Not only does tuberculin act in a harmless way in such cases, but it always does positive good.

If there be any tuberculous lesion the fever caused by the dose soon abates, and within a few days—often less than seven—there is an increase of weight and a subjective feeling of improvement which the patient spontaneously acknowledges. I admit that there is sometimes a severe reaction, 102° to 104°, even 105°, and therewith a temporary loss of weight. This uniform improvement in the general health

and increase in weight, which I have observed for a great number of years—at least ten years—is absolutely out of harmony with Wright's views upon the "negative phase." Not only this simple experience fails to give any support to Wright's opinion that reactions depress the state of immunity for ten to fourteen days, for I have observed, not in a few, but in scores of cases, that one can attain to the production of a high degree of immunity by *increasing* doses at far shorter intervals than ten to fourteen days.

CASES IN WHICH TUBERCULIN GAVE A NEGATIVE RESULT

1. Mr. G., 32.

Asthma, Asthma for thirty-two years. Physical signs of bron-
called chiectasis; no tubercle bacilli. 001cc., 005cc. and 001cc.
Phthisis. Old T. negative.

2. Mrs. M., 27. Mother of four children.

Called early Came from country (150 miles) to see Dr. F., who diag-
Phthisis nosed pulmonary tuberculosis and ordered to country.

Tested: 001cc. 005cc. 001cc. Absolutely negative.

No physical signs; purely gastric symptoms. Seen several years after; quite well.

3. A. B., 12.

Spinal Bedridden for eighteen months in country. Consulted
Curvature. a good physician in Sydney, who treated her for a month for caries, and then sent her to hospital. She was deadly pale, wasted to a shadow, with very marked lateral curvature, both knees fixed on abdomen, and it was impossible to extend them. I suspected "functional trouble," and tested.

001cc. Old T. 984; 005cc. Old T. 984.

01cc. Old T. 984. Not the slightest swelling in arm.

Straightened limbs under chloroform and fixed in splints. Then fed by nasal tube, and in less than six months patient walked into hospital, erect and in blossoming health.

4. Miss D.

Diagnosed as Seen by Dr. S. S., who diagnosed "incipient phthisis,"
Incipient in spite of the fact that I had tested her with tuberculin
Phthisis and obtained no reaction. No physical signs and no cough. The mother was placed in a dilemma. I suggested to her that if Dr. S. S. and I met in consultation, we might settle our differences. Dr. S. S. refused to consult with me. The mother followed my advice. Dr. S. S. had advised that the daughter should be sent away from home into the country for two years. The daughter remained at home, and has been perfectly well for six years.

5. R.

Weak chest. Dr. C. and Dr. McS. told him he had "a weak chest."
 Tested: 001cc O.T., 005cc. 01cc. *Negative.*

6. Miss R., 24, 7 st. 10.

Anæmia and loss of wt. Treated with creosote by Dr. F. who said both lungs were going.

Diagnosed as pulmonary tuberculosis. Tested: 001cc. Old T. 98'4".
 005cc. " 98'4".
 01cc. " 98'4".

No physical signs and no other evidence.

7. Mr. G., 35. Always husky.

Sent out from England for early tuberculosis, giving up good appointment. On arrival consulted me. Could find no physical signs.

Tested: 001cc. Old T. 98'4".
 005cc. " 98'4".
 01cc. " 98'4".
 01cc. " 98'4".

8. C.

Signs. Loss of weight.

Tested with tuberculin. *Negative result.*

9. Miss D., 27, 3 st. 9.

Cough eighteen months. Poor appetite.

Dr. M., diagnosed pulmonary tuberculosis, and ordered her to give up work and go to Hay or Bathurst, several hundred miles from her home (Lismore). Consulted me.

Tested: 001cc. Old T. 98'4".
 005cc. " 98'4".
 01cc. " 98'4".

Advised her to return home.

10. Mr. G.

Accident on harrow-field, resulting in pneumothorax. English doctor doubtful if any tuberculosis.

Tested: No reaction to tuberculin (01cc.)

11. Miss E.

Pulmonary Phthisis. Dr. M. treated for a year for pulmonary tuberculosis. I found swarms of various bacteria in phlegm and suspected atrial abscess. Small mass of pus in middle fossa of nose.

Explored sinuses with cannula, and washed out filthy sort of pus. Also tested. Quite negative. Even 01cc. Old T. 98'4".

12. H., 13.

Sent to country for lung disease. Cough for nine years. Much phlegm. Tested even 01cc. Old T. Had no effect.

13. Nurse H.

Nursed phthisical patient for eight months under a doctor, who apparently had not given any instructions about expectoration. Tuberculin up to 100cc. Old T. Negative.

14. G. W.

Diabetes insipidus (nineteen pites or more daily) and reflex paralysis to light in left eye, and also loss of reflex by cross-path.

Tested: 100cc. Old T. 98'4".

100cc. " 98'4".

100cc. " 98'4".

Accordingly tuberculous deposit excluded.

15 & 16. An anxious father, himself a medical man, brought his two "delicate" sons for examination. One son had already spent six months in a sanatorium, and was no better.

I tested both the boys, as I could not find the physical signs upon which the father made his diagnosis. Both boys failed to react to 100cc. Old T., much to the father's delight.

17. Mrs. S.

Advised by three doctors to go to a sanatorium where advanced cases are admitted, because she had married her husband, who had recently died (also her daughter, No. 26). I tested her with negative result.

100cc. Old T., 98'4".

18. Mr. F.

Rheumatism. Treated for pleurisy for a year. Could find no signs yet he had pain enough.

Tested. No reaction.

19. Miss L.

Professional musician.

Anxious about state of lungs.

Tested: 100cc. Old T. 98'4".

20. Nurse G.

Nursed case of advanced phthisis for long.

Cough, and "one or two spots" of blood.

Tested: 100cc. Old T. 98'4".

21. Miss C.

Always a cough, and much spitting.

Tested: 100cc. Old T. 98'4".

22. Nurse H.

Named "phthisical" case for eight months under a doctor, who gave no instructions about spittoon, etc.

Tested: 100cc. Old T. 98'4".

23. H., 13.

Cough for nine years; much phlegm. Sent to country. No hemoptoe.
Tested: Absolutely negative.

24. M., P.

Said to have "pleurisy." Found no signs.
Tested: 10cc. Old T. 98.4.
Anxious, because he had a fine tenor voice.

25. Miss L.

Anxious about her lungs.
Tested: 10cc. Old T. 98.4.

26. Miss S., daughter of No. 17.

Long exposed to infection.
Tested: 100cc. Old T. 98.4.

27. Miss U., 16.

A chronic ulcer on knuckle—might be tuberculous or specific.
Tested: 10cc. Old T. 98.4.
Healed under simple treatment.

28. Mr. S.

Thought he had lung trouble. Sent by leading chemist.
Tested: 10cc. Old T. 98.4.

29. A. R., 12.

Temperature 99.6°. Pulse, 115.
Tested: 10cc. Old T. 98.4.

30. C. S., 4.

Subacute synovitis.
Tested: 10cc. Old T. 99.

31. Mr. G.

Anxious about state of lungs.
Tested: No reaction.

32. Nurse B. In 1904.

Treated with creosote by Dr. F., who said both lungs were gang. Also anemia and loss of weight.

Tested: 10cc. Old T. Negative.

Saw this lady to-day, after lapse of five years. Quite well (1909).

33. Mr. B., 18. 10 st. 10.

Sent to me for "pulmonary tuberculosis"—hemorrhages. Several crises, in January, 1907, and in seven weeks had six hemorrhages. No Th. in sputum.

Tested: Absolutely negative to tuberculin (10cc.)

Diagnosis: *Hypertrophic disease of lungs*.

Sent to surgeons, who had sent patient to me.

34. Miss L.

(Signs at L. apex) depression and deficient movement. Anxious, because she was going to be married.

Tested: wacc. Old T. Negative.

35. Miss H.

Treated for 14 years in sanatorium for phthisis; had *hemoptæ*. Typical initial stenosis, and no signs of tuberculosis in lungs. I sent her to leading physician, who confirmed my opinion. Was about to be tested, but afterwards saw no object in it.

36. Mr. M., 20. 9. 11. 4.

Liable to bronchitis.

Tested: No reaction to wacc. Old T.

37. Miss M.

Exposed to infection.

Tested: No reaction. Mind much relieved.

38. B.

Tested in hospital. Negative.

39. S.

Tested in hospital. Negative.

40. E. B.

Tested in hospital. Negative.

41. Miss C.

Anxious about her lungs.

Tested: No reaction.

42. Mr. H.

One sister died of phthisis.

One sister in Stage I. treated by me.

Tested: No reaction to wacc. Old T.

43. E. R.

Hysterical hemiplegia.

Tested: No reaction to wacc. Old T.

44. P. G. C.

Bronchitis.

Tested: Negative.

45. S.

Cystitis and pyelitis.

Tested for Dr. M. No reaction.

46. C.

Gonorrhœal arthritis.

Tested: No reaction.

47. —

Simple arthritis.

Tested: No reaction.

48. C.

Cystitis.

Tested: No reaction.

49. O. K.

Asthma.

Tested: No reaction.

50. Miss M.

Sister died of phthisis.

Tested: No reaction.

51. Ch.

Anxious about lungs.

Tested: No reaction to 'vacc. Old T.

52. Mrs. W.

Sister treated for early tuberculosis.

Tested: No reaction to 'vacc. Old T.

53. Mr. R.

Hæmorrhages from lungs—recurring. Crepitations below right nipple.

Tested: Absolutely negative to two doses of 'vacc. Old T.

Diagnosis: *Hydatid of the lungs*.

X-rays showed a shadow. Sent to surgeon, who opened chest, but could not locate cyst.

Three months after coughed up cyst.

54. B.

Sent by Dr. H. H. I examined blood and found marked increase of lymphocytes. Symptoms chiefly abdominal. Suggesting chronic disease of pancreas. Put in hospital and feces and urine examined. No evidence of disease of pancreas.

Then diagnosis, probably tuberculosis or syphilis. Negative reaction ('vacc. Old T.).

Anti-syphilitic treatment cured him of all symptoms.

55. Miss D.

Sister treated for tuberculosis.

Tested: 'vacc. Old T. No effect.

56. Miss P.

Had had severe nasal trouble with obstruction and asthma. Removed obstruction.

Anxious about lungs, as she was going to be married.

Tested: 'vacc. Old T. No effect.

57. *Case 115* (included at the end of records of Stage I, p. 176, of printed matter) is a striking instance of the pre-eminent value of tuberculin in diagnosis.

In December, 1907, I wrote: "Case 115 is one of the most puzzling cases I have ever seen. She came from Turin. These two doctors diagnosed: *Tuberculous disease of the knee-joint*; and patient then came to Sydney and saw a surgeon, who confirmed the diagnosis. I then saw patient, and was of the same opinion, and proceeded at once to treat her, thinking that the nature of the case was certain. However, after a few doses of P.T.O., which, though rapidly increased, produced no effect at all, I began to be sceptical as to the existence of tuberculosis. I therefore gave some doses of Old T. up to 100,000, with no effect in arm, or joint, or in system. I then applied Calmette's ophthalmic reaction, which was twice absolutely negative." At this stage I told the mother I could no longer believe it was tuberculosis, and another surgeon met us in consultation. He had already got the knee under X-rays. Dr. C. A., a distinguished surgeon, and Dr. Harris, our first radiographer, decided that it was tuberculosis. There were five good opinions against mine. I went on to say: "Patient is still having 'tuberculin' (because I knew it could do no harm), though I fear the possibility of *myeloid sarcoma of the lower end of the femur*. It may be a chronic inflammation of the knee-joint, not tuberculous. There was no history of injury and no pain at first." Then I left Sydney.

Two months later Dr. Maitland, who had charge of the case, wrote: "I took Miss B.'s leg off this morning. I had another X-ray picture taken which brought out the fact that there was sarcoma of the lower end of the femur. I sent the case to Dr. Harris in the first instance, telling him that it was probably sarcoma, but he assured me that there was no *corpus* and that it was one of the clearest pictures he had ever taken, and asked him again six weeks ago, but he assured me that it was no use; he was sure *there was no sarcoma*. This time, however, there was no doubt.

"Yours truly,

"H. L. MAITLAND."

Could there be a clearer vindication of the value of tuberculin in diagnosis? My opinion based on the negative reaction with tuberculin was quite right—surely a most instructive lesson for all of us.

Many other cases of a similar kind have submitted to the simple test, and in no instance was the patient affected by the dose, and in all cases the patients were gratified at learning the fact that there was no tuberculous disease of the lungs—in such a simple way. In my experience it needs but a little persuasion by one who is confident of the enormous value of the method to exploit this marvellous dis-

covery of Professor Koch. I have not yet met any condition that contra-indicates its use according to Koch's original directions.

TUBERCULIN AS A MEANS OF CONTROLLING RESULTS.

Tuberculin is also of very great value in determining the effect of treatment, especially of specific treatment. No matter what methods we use, we have a very stubborn foe to face. Disappointments in the form of relapses are of constant occurrence. The disease may be stayed for a year—for two years and longer—even for ten years, and then reassert itself—a sure sign that the bacilli have been "scotched, not killed." The important question then arises whether we can detect this recrudescence in a very early stage, so as to strike the enemy again when he is beginning to awake and ready to manifest further activity. Sometimes indeed this further activity shows itself in riotous fashion, especially when a mixed infection with influenza bacilli, or streptococci or pneumococci is engrafted upon the original tuberculous infection. Again and again I have witnessed and proved the very existence of this complication, and oftentimes it is the mixed infection which intensifies the trouble and leads to the exhaustion and death of the individual.

At this stage especially (as specific treatment has not yet been evolved for these mixed infections) open-air treatment and sanatorium methods may help us in our serious task, and the tubercle bacilli may then again settle themselves down to sleep for a season. In all such cases it is wise to try and keep the tubercle bacilli enchained in their fibrous envelope until the bacteriolysins can complete the destruction of the bacilli. Accordingly it is a part of my plan to test all patients who have been already treated with tuberculin at least once a year, using as test doses larger doses than those used as a first test. My plan is to give at intervals of two or three days, .005cc., .01cc., .03cc., .05cc., and .1cc. of Old Tuberculin. If a reaction follows, I always advise a further course, and often one can proceed up to 100 P.T., or 2cc. T.R., or 3 or 4cc. T.E. *within two or three weeks*

without any disturbance at all. As a rule, if a course of P.T. up to 1cc. has not eradicated the disease, I begin at once with the tuberculins made from the human strains (Old Tuberculin, T.R. and T.E.). Quite recently I have been using the special T.A.F. (tuberculin albumose free), but I am not in a position to say whether T.A.F. should entirely, or at all, replace Old Tuberculin.

CHAPTER III

PROGNOSIS

IT is extremely difficult to estimate the effect and value of different methods of treatment in pulmonary tuberculosis, because this disease runs such a variable course. As I have said elsewhere, the tubercle bacillus may lie in ambush for months and years, and in this latent stage of the disease the health, the strength, and the appearance may suffer but little. The disease may become thus quiescent at any time; it may relapse after months or years; it may reach a serious stage, involving the greater part of a lobe and end in perfect restoration to health. In spite of treatment of any and every kind, the disease may never relent, closely pursuing the victim till death. Without any treatment the disease may come to a standstill, and never again disturb the health. The most affectionate regard for logic may not restrain us from attributing success to our own small efforts and failure to the vagaries of the disease. In such diseases as diphtheria, typhoid fever, pneumonia, etc., we can attempt to measure the efficacy of methods by the number of lives we save in every hundred cases compared with the number of lives saved by other methods. Even such a comparison is open to the serious objection that in different epidemics the virulence varies greatly. Still a method that uniformly lowers the rate of mortality is worth trying. In such diseases the method succeeds or fails in the course of a few days, or at most a few weeks—so that we are not left long in doubt. Either our method succeeds or fails. *There are no partial successes.* The disease either comes to an end or death occurs. In pulmonary tuberculosis it is literally impossible to trace the infection to its source with scientific accuracy, and it is

equally difficult to fix the time of its occurrence. Even when we find that the disease exists, we cannot forecast the likely course the disease will take. Some authorities state that the average duration of the disease is about four or five years; others say seven or eight years. These calculations based upon old hospital records are not trustworthy. It does not follow that, even though pulmonary tuberculosis has been the cause of death, this disease and no other has been the actual disease from the outset. Pulmonary tuberculosis may engraft itself upon other morbid states of the lung or body. Indeed when we cannot control our observations, we may take too much for granted and arrive at unsound conclusions. With a full consciousness of the uncertainty of records dating back several years in the lives of patients, I have attempted to estimate the duration of all cases that have come to me in a late stage (Stage II-III and Stage III). We need not stay to consider how long the disease may exist before the symptoms attract attention. Von Behring thinks the disease, even pulmonary tuberculosis, is sown in infancy. It is likely that six months at least are required for the development of the symptoms and signs of pulmonary tuberculosis; how much longer we have no means of knowing. Even medical men fail to recognise the disease in the early stages. As an illustration, Case 104, Stage I, consulted three doctors. One said there was "no mischief in the lung"; another said there "had been disease, but it had been arrested"; while the third said "there was mischief." Of three medical men, one was right, as I proved by means of tuberculin. If medical men cannot recognise the disease, how can we expect patients to tell us accurately when the disease began? From the analysis of late cases I should certainly say that in the majority of cases the disease runs its course in three years or less. Hence in testing the merits of any system of treatment, especially in the earlier stages of the disease, records, that do not allow the lapse of two or three years after the system of treatment has been put on trial, are valueless. For this reason one must put aside the evidence of any records of sanatoria, unless these records have been controlled by after examinations. All Wright's records are stamped with the

same radical defect. In many of Wright's records, treatment was adopted early in one year, and the successful results were claimed—some within the year—most of them within two years after the adoption of treatment. Wright did not himself report any cases of pulmonary tuberculosis successfully treated, but suggested that early fever-free cases might be treated with success. He left others to exploit his opsonic method in pulmonary tuberculosis, and so far no trustworthy evidence has been forthcoming. The cases have not been controlled by time, and as Wright and his followers ignore tuberculin as a diagnostic agent, they would hardly approve of its use as a means of testing the value of the opsonic method. Further, Wright and his disciples have so far not attempted to seriously *test the value of the opsonic method of treatment in the second and third stages of pulmonary tuberculosis*, which nowadays constitute the great majority of cases that come under the notice of the physician. There can be no doubt that sometimes tuberculous lesions show an extraordinary latency. Perhaps sometimes their latency is due to an attenuated virus, sometimes to the quality of the germ (bovine type), sometimes to the resistance of tissue. But this latency is a feature mainly of the early stages. It is not often that we flatter ourselves or the unfortunate victim with the idea that *latency is a striking feature of the second stage*. Real latency in the second stage rarely lasts even a year. In Stage II-III, or Stage III, I have never seen latency. In like manner the "healed tuberculosis" of the post-mortem room is essentially a lesion of the first stage. *How often do anatomists find in the dead body healed lesions of the second stage?* At any rate, I can only remember one or two instances in which I found at the post-mortem examination that a tuberculous lesion of the second stage of pulmonary tuberculosis had even come to a standstill—was no longer progressing. I have never seen a lung in such a condition that I should have inferred that at one time there had been the solid areas of tuberculous disease of the lung characteristic of the second stage, and subsequently the process had regressed and the lung had healed. Tuberculous formations cannot easily disappear and leave no trace of their existence.

in this respect contrasting with the inflammatory formation of croupous pneumonia. If such healing of a tuberculous lesion of the lung in the second stage of pulmonary tuberculosis is rare, healing of the lesion in the third stage must be a curiosity. The gravity of the prognosis in pulmonary tuberculosis increases not in arithmetical, but in geometrical proportion with the stage of the disease. Accordingly, cases in the second and third stages, which mostly progress and exhaust the patient within two years, may be used to gauge the relative merits of different systems of treatment. So many early cases heal spontaneously (post-mortem evidence), so many remain latent even for years, that we cannot say for certain that any remedies we use have arrested the disease. In the second and third stages it is otherwise. Rarely does the disease, when it has reached the second stage, regress or come to a standstill. Accordingly, if after treatment the disease is arrested and does not become again active for two or three years, the treatment has done good, and the beneficial effect of treatment may be measured by the number of years that the disease remains in abeyance. Moreover, it is obvious that any method of treatment that then acts beneficially when the disease is advanced must of necessity benefit also those affected with disease in a less advanced stage. It is also possible that a method which benefits those in an early stage may be of little or no value in the later stages. However, I wish to urge that a satisfactory comparison of the relative merits of different systems of treatment demands that we should consider the effect of treatment in the different stages, and especially in those stages when the prognosis is relatively unfavourable. *The more unfavourable the prognosis the greater the virtue in the method that succeeds in prolonging or saving life.* For this reason I would direct special attention to the records of cases obviously in Stages II., II.-III., and III., treated exclusively by means of various forms of tuberculin, let me add, in very large doses and with a total disregard of Wright's Opsonic Index.

In the appended list (see Appendix) I have given all the cases in late stages that have come under my notice within the last few years, and the average duration of the disease is

about three years. Out of 180 cases about 60 cases were in a hopeless condition in less than one year, 60 in less than two years, whilst the remaining 60 (one-third) had reached the hopeless stage in periods ranging from three years to ten years or more—the majority of this (third) within three years. All these cases were hopeless when I saw them. Many of them had had open-air treatment, climatic (mountain air) treatment; most of them, alas, had been drugged with creosote, with the result that in less than three years they were dying. I might in justice call all these cases "control cases," illustrating the failure of other methods of treatment. Is it a mere chance that while in the hands of other men scores of cases passed from the first into the II–III or III stage in three years, not a single case can be recorded against me in the last six years, in which under tuberculin treatment the disease has passed from the first to the second stage?

The following extracts from notes are significant:—

"Saw Dr. — one-and-a-half years ago, who said 'nothing the matter with lungs.' Then saw Dr. —, who snipped off uvula for cough. Sent to country by several doctors. Now in late stage."

Again: "Seen nine doctors. A year ago Dr. — (a good doctor, but no believer in tuberculin) said lungs were sound. Had open-air treatment. Weight has been good, but disease has advanced to Stage III."

Again: "Nine months in sanatorium—sent out 'lungs dry and quiet,' yet now in Stage III."

Again: "Treated for indigestion for nine months; then sent up to country. Stage III, in less than one year after."

Again: "Patient in Stage III. Milks cows."

Again: "Saw Dr. S—, who said it was 'all in the throat'—a year after in Stage III."

Again: "Came from New Zealand three months ago and gained 14 lbs.—yet now (three months later) in Stage III. Pulse 140."

Again: "Pleurisy twelve months ago—sent to country—died in another six months."

Again: "Dancer, nine months ago weighed 9 st. 4 lbs.

Had 'pleurisy.' Dr. S—— said 'nothing the matter with lungs'—less than a year Stage III."

Again: "Pleurisy fifteen months ago—now in Stage III."

Again: "Saw Dr. R—— one-and-a-half years ago. He said 'chest was weak'—open-air treatment. Now hopeless in Stage III. c.tuberculous laryngitis."

Again: "Saw Dr. C—— a year ago—sent to Bathurst, gained weight and lost cough—in Stage III, and died three months after."

I have records of about 200 cases in which ordinary methods have utterly failed within two or three years for the most part. With tuberculin I have had no single failure of this kind. Almost all these cases had had ordinary treatment—had been sent to the mountains and well fed—yet in less than three years the greater number are dead. These numerous cases show the uselessness of haphazard trips to the country. Many of these patients had the early warnings—pleurisy, throat symptoms, cough, and loss of weight; and if tuberculin had been used their fate would have been very different. By ordinary methods these cases in Stages II, III, and III, can hardly live one year—by means of tuberculin many of them may live in apparent health for many years.

With improvement the cough lessens and ceases, phlegm ceases, tubercle bacilli are not found, energy increases, and often there is also gain in weight and the appearance of health. But lest the enemy may awaken it is best to test all such cases from time to time, once in six months or once a year, so as to be able to give further treatment with tuberculin if there is any indication of the enemy becoming active again. A reaction with tuberculin in doses 5 mgrs., 15 mgrs., 30 mgrs., and 50 mgrs. is such an indication. If the treatment has not been by means of tuberculin, tuberculin may also be used for the same purpose, but in the smaller doses used for the original test. In Stage II, the prognosis is quite good when tuberculin is used for treatment and at intervals afterwards to control the result. The danger is that the patient, after being well for several years, may think he can dispense with the testing by means of tuberculin. That over-confidence may be fatal. It is a remarkable record that of all

the cases in Stage II. treated with tuberculin since 1902 there has been but one death, and that was due to this over-confidence. He relapsed nearly six years after treatment. My statistics show successes to the extent of over 90 per cent. Allowing for possible luck, I have little doubt that in Stage II. 70-75 per cent. of successful results can be obtained and maintained by means of tuberculin at least for many years. In Stage II.-III. I have secured as good results with tuberculin as can be obtained through sanatoria in Stage I. My records show quite 60 per cent. of permanent results at the end of 1907. Of course, when one knows the extent of the disease, it is little short of marvellous that these victims should now be enjoying apparently good health.

In Stage I. I agree with Professor Koch that pulmonary tuberculosis "can be cured with certainty by means of tuberculin." If I gave only the records of cases, numbering at least a hundred, which I am able to control after a full course of treatment, I should have to give a record of 100 per cent. successes. At least, I feel sure that by patience, courage, and the intelligent use of tuberculin in diagnosis, prognosis and treatment infinitely better results can be obtained than by any of the so-called rational methods that are now in fashion. I am convinced that the degree of immunity depends upon the amount of tuberculin that can be given without causing any reaction, and accordingly I utterly repudiated Wright's views and methods when he published them, and clearly proved that in pulmonary tuberculosis the opsonic index could not help one and might easily lead one astray. Medical men in Sydney imagined that I attacked Wright's system from mere perversity, but the point at issue for me was a very simple one, as I had an experience that left me no option in the matter. I had used tuberculin in the treatment of pulmonary tuberculosis with great success, and I found that the larger the doses were, the more permanent the results. When, then, Wright appeared as the advocate of tuberculin in minute doses without even trying it in a single case of pulmonary tuberculosis and his own enthusiastic disciples entered the field as the new-born advocates of tuberculin treatment in pulmon-

ary tuberculosis, it was time for me to step into the arena and tell these mesmerised disciples that tuberculin in homoeopathic doses according to Wright's opsonic system had not been proved to be of any value at all, and that, in fact, I had proved that tuberculin in large doses was an extremely valuable remedy in pulmonary tuberculosis. The logic of Wright's disciples in Australia left them open to attack, and lest the failure of Wright's system might again put back the clock and cause tuberculin as a remedy to be again discredited, it was my bounden duty to express the conviction that Wright's system applied to pulmonary tuberculosis must inevitably lead to failure. My own successful results were due to large doses of tuberculin, and I had proved the value of large doses in hundreds of cases; Wright's system was essentially theoretical and problematical as applied to pulmonary tuberculosis and entirely incompatible with my practice, which had been very successful. At a demonstration of Wright's extremely ingenious technique for determining the opsonic index, the author failed to estimate the index, and afterwards I merely asked the question, "Is it worth while?"

We may now proceed to consider whether it is worth while to follow the method of treatment which Sir Almroth Wright has evolved from his studies relating to the opsonic index.

TUBERCULIN AS A GUIDE TO PROGNOSIS AFTER SPECIFIC TREATMENT

It is quite certain that even when large doses of tuberculin have circumscribed or neutralised the power and activity of the tubercle bacilli, the bacilli may slowly regain energy and strength, especially if some complication, such as a secondary infection, weakens the resistance of the tissues. Accordingly the strength of the enemy must be reconnoitred from time to time. Else virulent and active bacilli may be let loose in consequence of the severe tissue-changes of acute inflammation, and the tuberculous process makes a fresh start. This is far more likely to happen when the production of antibodies has not been ready and abundant. To my

mind, it is hardly conceivable that the system of dosage advocated by Wright according to his epineuric method—repeating very small doses, without increasing them, at long intervals of time—can lead to a ready and abundant production of antibodies at such a crisis. If this system succeeds, it is hard to see why small doses, which must be absorbed from the tuberculous focus during the progress of the disease, do not create immunity. In diphtheria and tetanus the dose sufficient to establish immunity in a normal animal is smaller than the dose necessary to arrest the disease, when once it has established itself; and in order to create a high degree of immunity in a horse by a process of active immunisation it is necessary to increase the doses of the diphtheria-toxin to very large doses. In the treatment of tuberculosis by means of tuberculin the process is also one of active immunisation. But in tuberculosis there is of course something more than the production of a toxæmia by means of a toxin; there is also the resistant tubercle bacillus, which contains an endotoxin in its cell-substance, and there can be no certain arrest of the tuberculous process until the tissues acquire the faculty of quenching the life of the bacillus itself. The tissues may, and often do, check the activity of the bacillus and prevent the absorption of the soluble toxins by undergoing fibroid organisation and forming around the colony of tubercle bacilli a more or less dense and impenetrable wall. Yet until the bacillus is killed—and it may be found alive in calcareous masses (Rahinowitch)—one dare not speak of arrest of the disease in a scientific sense. This is the rock upon which so many of us are constantly making shipwreck. There is no absolutely certain way of telling when the tubercle bacilli are dead or even inert. By the very best methods, including the use of tuberculin, we may hardly avoid error. If we do not use tuberculin in the attempt to learn whether there are still living and active bacilli in the tissues, we must experience those rude awakenings that are thrust upon us, when a severe hemorrhage or a mixed infection supervenes. In order to thwart the enemy I hold that we should vigorously train the tissues to become efficient producers, not merely of antitoxic,

but also of bacteriolytic substances, and in my opinion powerful stimuli in the form of large doses of tuberculin, not once or twice, but many times, may be needed to cause the dissolution and destruction of the tubercle bacillus closely enveloped in its dense if not impenetrable armour of waxy material. By Wright's method of small doses—not increased and at long intervals of time—the stimuli are always weak, and even though the opsonic index may be raised somewhat, the tubercle bacilli may still revel in the tissues, and at a later period play sad havoc both locally and in the general system. Certainly when a complication such as a secondary infection occurs, the tubercle bacillus will make the most of its opportunity unless there are powerful antibodies to hold it in check. For this very reason we must keep the army of leucocytes in such excellent training that the tubercle bacilli cannot spring into activity even when a mixed infection lowers the resistance of the tissues. This can be best achieved by keeping a constant eye on the tubercle bacillus and testing its vitality. Thus the use of tuberculin may postpone or prevent a serious crisis. If a ready reaction to tuberculin occurs, fresh activity on the part of the bacillus has begun or is imminent. I have invariably found that whenever a fresh attack is threatening, extreme sensitiveness to tuberculin has returned, and unless this extreme sensitiveness is recognised early and dealt with promptly a mixed infection may supervene and prevent the use of tuberculin as a curative agent. Accordingly we must always be on the watch for this over-sensitiveness to tuberculin, and there is but one way of detecting it. Unless we use tuberculin from time to time in all cases, *even when the most satisfactory results have been obtained by large doses of tuberculin*, we are not acting with circumspection or prudence. By thus exploiting tuberculin we may keep the disease constantly "in check," with a view to ultimate check-mate; and this use of tuberculin is another step towards the successful treatment of pulmonary tuberculosis. In all those cases of mine in which a relapse occurred with a fatal result, a mixed infection established itself and was the essential cause of exhaustion and death. So long as the mixed infection

is kept at bay, the tuberculous process can be dealt with by the specific remedy. As soon as the mixed infection gets the upper hand obviously tuberculin fails, because it can have no effect upon the mixed infection, and in some instances it may do harm by encouraging the inflammatory changes in the tissues, which favour the virulence of the organisms associated with mixed or secondary infection. Then especially is it necessary to make haste slowly, and no attempt should be made to use large doses of tuberculin or to increase the doses rapidly. In these cases a careful analysis of the sputum for the purpose of determining the nature and extent of the mixed infection is a *vincit quod non* of specific treatment. As a rule, if the tuberculous process be early, the mixed infection will clear up and then specific treatment can be adopted with every hope of success. In other cases it may be necessary to deal with the mixed infection by the open-air method and treatment in the sanatorium. The mixed infection may yield to these measures in three or four weeks, and then specific treatment can be adopted. In the later stages (II. and II.-III.) mixed infection often takes firmer root and causes more serious symptoms and greater destruction of lung tissue, even to cavitation. Then it may be very difficult to uproot the cause of the mixed infection, and by slow degrees the patient passes into a parlous state. During the year of my absence in England this was the sad fate of three of my patients who had responded to tubercula treatment in the most satisfactory way. Two of them had been free from all symptoms for nearly six years. The other case had a severe septic abscess of a tooth and a fatal septic infection of the air passages supervened. The lesson to be engraven on one's mind from this experience is the paramount importance of testing all cases that have been under treatment at regular intervals of time—certainly once a year. In two of the three cases above recorded I have little doubt that if the tests had been made every year these calamities might have been avoided. And why should one not test in this way? The test has absolutely no effect if the disease has been arrested—no effect at the site of injection and causes no rise of temperature or other symptom. If there is

any reaction, I give a further course. The test must be carried out in a more energetic way—by larger doses—than when one tests in the first instance. Instead of giving doses .001cc., .005cc., and .01cc. T.A. I give .005cc., .015cc., .03cc. and .05cc. or .1cc. Since my return I have tested several cases; two of them gave slight reactions. I gave a further course without the least trouble. *A very satisfactory feature in these cases that still react a year or so after a course of tuberculin treatment is that one can proceed to a full dose (1 cc. P.T. or 2 cc. T.R.) within two or three months without any reactions at all.* This alone is surely equivalent to an indictment of Wright's opsonic system.

APPENDIX

CASES ILLUSTRATING DURATION OF DISEASE.

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| 3 years. | 1. A. B. C., 30. 3 st. 12.
Stage III. in 3 years. Temp. 100°; 101°. Pulse 105. |
| 1 year. | 2. Miss C., 35.
Stage III. in less than a year; also ulceration of larynx.
Cough for four months. |
| 3 years. | 3. Mrs. D.
Stage III. in 3 years. "Terrible hæmorrhages." |
| 8 years. | 4. Mrs. E.
Family history.—Father 60 when patient 5 years old.
Brother 2 years older, 40-52. Two sisters 40-46 and 46-48.
Patient nursed sister 16 years ago and slept with her,
married brother 10 years ago, patient had hæmorrhage
3 years ago—5 years ago a severe hæmorrhage (O)—
called bronchitis, and influenza—went to Madge, then to
Rushurst (3 months), then Mt. Victoria (3 months); then
sanatorium (fomentations); rubbing with salt and castor-
oil (vegetarian diet), "promised to cure." Took
guaiacal for 2 months—caused retching—gave it up.
5 years ago became hoarse, saw specialist (Dr. B.), who
said there was nothing the matter. Now Stage III. with
tuberculous laryngitis. |
| 8 years. | 5. S. P. B., 31. 10 st.
Stage II.-III.—c. tuberculous laryngitis, 3 years. |
| 1 year. | 6. C., 32.
Stage III.—c. laryngeal tuberculosis, 8 months. |
| 1 year. | Several doctors advised change; went away, came
back worse and very hoarse. |
| 2 years. | 7. D.
Family history very bad.
Stage III. in 2 years; died in 6 months. |
| 1 year. | 8. W. A., 31. 10 st. 9. sister 40.
Stage II.-III. in 1 year. |

- 2½ years. 9. Miss B., 26.
Stage III. in 2½ years; died in 4 months.
- 1 year. 10. C. P., 14. 5 ft. 8.
Stage III. (three doctors said "dying")—9 months II, and in Stage III. was having physical culture.
- 4 years. 11. Miss L., 34.
Stage II-III. in 4 years.
- 3 years. 12. W. G., 35.
Stage III. in 3 years; died in 6 months.
13. W., 35. Waiter.
Stage III.—began 3 months ago.
- 2 years. 14. W. D., 35.
Stage II, III. in 1 year and 10 months.
- 1 year. 15. Mrs. H.
Stage II-III.—c. tuberculous laryngitis—1 year (child while bathing a year ago).
- 1 year. 16. Mrs. R.
Stage II-III. in 1 year.
- ½ year. 17. Miss C., actress, dancer, 20. 7 ft. 8.
Stage III. in 3 months.
- 4 years. 18. Mrs. McW.
Stage II-III. in 4 years.
- 1½ years. 19. Mrs. McC., husband 80 1½ years ago.
Stage III.—in 1½ years tuberculous laryngitis.
- 3 years. 20. Miss R. H.
Stage II-III. in 3 years.
- 1½ years. 21. Mr. L., 12. 8 ft. 4.
Stage III., pleurisy 15 months before.
- ½ year. 22. Miss S., 23. 5 ft. 2.
Stage III.—3 months ago influenza.
- 1½ years. 23. W., 42. 11 ft. 4.
Stage II-III.—c. fatal more than a year. "Fourteen days after operation for double by Dr. McC. had hæmoptysis.
- 2 years. 24. Miss B., 26. 7 ft. 4.
Stage III. in 2 years.
- 3 years. 25. Mr. P., 35.
Stage III.—c. tuberculous laryngitis in 3 years.
- 3 years. 26. Miss Y.
Stage II-III. in 3 years.

27. Miss P., 25. 8 st. 7.
 ½ year. Mother 46; brother 46.
 Stage II-III in 6 months.
28. Miss E., 20. 2.
 1 year. Stage III—cough last winter.
29. C., 31. (Hospital.)
 1 year. Pulmonary tuberculosis and tuberculous meningitis and myelitic cord in 10 months; began as pleurisy.
30. S., 41. 14 st. 4.
 1 year. Stage II. c.—tuberculous laryngitis in 3 months; died in 6 months.
31. Miss T.,
 2 years. Stage III in 2 years.
32. M. P., 18.
 3 months. Stage II-III in 12 weeks after pneumonia.
33. Mrs. D. Father, 90; mother, 70; mother's mother,
 4 years. 84. Yet on husband's side, as follows: Husband's sister married, her husband 46; another sister married and her husband 46. The husband married patient, and she is in Stage III in 4 years. Began as pleurisy, and treated by Drs. S. M. H. K. and McM. (j).
34. Rev. F., 31.
 ½ year. Stage II-III. or later—yet said to have begun 4 months ago.
35. H., 26.
 1½ years. Awful family history. Sister nursed mother in late stage, and in 6 months 46. Eldest of family 46 in 1895; 7 months later, Willie, third son 46, 1895; and last is in Stage III. All these cases began with hæmorrhage.
36. L. N.
 4 years. Reacted to tuberculin in P. A. Hospital, but refused tuberculin treatment. In 4 years in Stage III.
37. H., 28.
 3 years. Stage III in 3 years.
38. Miss A.
 8 years. Stage II-III—c. tuberculous laryngitis after many years; 8 years before pleurisy; kept alive for 7 years with tuberculin.
39. S.
 4 years. Stage III in several years.
40. MacN., 29.
 2½ years. Stage III in 2 or 3 years.

- 3 years. 41. Nurse H., 24.
Stage II.-III. in 3 years.
- 3 years. 42. Mr. P.
Stage III. in 3 years.
- $\frac{1}{2}$ year. 43. F.
Stage III.—"six months ago quite well."
- 2 years. 44. A. S.
Stage III. in less than 2 years.
- 2 years. 45. Capt. Y.
Stage II.-III. in 2 years.
- 3 $\frac{1}{2}$ years. 46. Mrs. S., 25.
Stage III. with extensive ulceration: 80 within 2 years
(28 months).
- 2 years. 47. D. T., 16.
Stage II.-III. in 2 years.
- 1 $\frac{1}{2}$ years. 48. Mr. M., 37.
Stage II.-III.—c. tuberculous laryngitis in 1 $\frac{1}{2}$ years.
- 2 years. 49. Mr. G.
Stage III. in 2 years (pleurisy 2 years before).
- 6 years. 50. Mr. B., 38.
Stage III. in 6 years.
- 1 $\frac{1}{2}$ years. 51. Miss C., 28.
Stage II.-III. in 18 months.
- 1 $\frac{1}{2}$ years. 52. Miss G.
Stage II.-III. in 1 year and 3 months.
- $\frac{1}{2}$ year. 53. Mr. C., 26.
Stage II.-III. in 6 months.
- 4 years. 54. Mr. P., 24.
Stage III.—c. uncertain of larynx in 4-5 years.
- 3 years. 55. Father O.F.
Stage II.-III. in 3 years.
- 3 years. 56. Miss G.
Stage III. in 3 years.
- 6 years. 57. Mr. D.
Stage II.-III.—(6 years ago severe hæmorrhage; pleurisy
5 or 6 years ago).
- 9 years. 58. Mrs. P.
Stage II.-III. in 9 years.
- 1 $\frac{1}{2}$ years. 59. Miss G.
Stage III., and ulceration of larynx in less than 18 months.

- 2 years. 60. Miss L. McM., 27.
Stage II.-III.—c. laryngeal tuberculosis in 2 years.
- 1 year. 61. Miss G.
Stage III., and death within a year.
- 2 years. 62. Miss E. R.
Stage II.-III.—c. laryngeal tuberculosis in less than 2 years.
- 5 years. 63. S.
Stage III. in 5 years.
- 4 year. 64. Mr. I. P. S.
Stage II.-III.—c. ulceration of larynx—3 years cough, expectoration, but he says "only six weeks."
- 3 years. 65. Miss G., 23.
Stage II.-III., "hemorrhage 3 years ago."
- 1 year. 66. Miss B., 36.
Stage III. in 1 year.
- 1 year. 67. Mrs. P.
Stage III. in 1 year.
- 1½ years. 68. Miss R. H.
Stage II.-III. in 1½ years.
- 3 years. 69. Mrs. B., 37.
Stage II.-III. in 3 years.
- 2 years. 70. Miss F.
Stage III. in 2 years.
- 3 years. 71. Mr. M.
Stage II.-III. in 3 years (pleurisy).
- 1 year. 72. Mr. W.
Stage II.-III. in less than a year.
- 2 years. 73. Miss B.
Stage III. in 2 years.
- ½ year. 74. Mr. K., 30.
Stage II.-III. in 9 months.
- ½ year. 75. Mrs. C., 29.
Stage III. in 10 months.
- 3 years. 76. M., 21.
Stage II.-III. in 3 years.
- 2 years. 77. G., 21.
Stage III. in 2 years.
- ½ year. 78. Mrs. O.
Stage III.—c. tuberculous laryngitis in 2 months.
- 6 years. 79. M., 31.
Stage III. more than 5 years.

2½ years	80. S., 19. Stage III. in 3 or 4 years.
1 year.	81. B. Stage III. in 1 year.
1½ years	82. Mr. R. Stage III. in 1½ years.
5 years	83. Mr. L., 37. Stage III. in 5 years.
1 year.	84. B., 18. Stage II.-III. in a year; died a year later.
3 years	85. B. Stage III. in 3 years.
1 year.	86. Mrs. B., 26. Stage III. in less than a year.
1 year.	87. Mrs. H. Stage II.-III. in less than a year.
3 years	88. H., 47. Stage III. in 3 years at most.
2½ years	89. Mr. H., 23. Stage II.-III. in 2½ years.
3 years	90. Mr. M. Stage III. in 3 years.
2 years	91. B. Stage II.-III. in less than 2 years.
2 years	92. M. Stage III. in 2 years.
3 years	93. Miss R. Stage II.-III. cough for years.
4½ years	94. Mrs. W. Stage II.-III. in 4½ years.
½ year.	95. Mrs. C. Stage II.-III. cough for 4 months.
1 year.	96. Mrs. L. Generalised tuberculosis in less than a year.
3 years	97. Mrs. C. Stage II.-III. in 3 years.
2 years	98. Miss L. Stage II.-III. in 2 years.
2 years	99. Miss C. B. Stage II. in 3 months, and dead within 2 years.
½ year.	100. Miss L. Stage II.-III. within 6 months.

- 5 years. 101. Miss MacK., 24.
Stage II.-III. in 5 years.
- $\frac{1}{2}$ year. 102. N.
Stage III.—9 months under Dr. C. F. K. Also tuberculous laryngitis and fistula; dead within a year.
- 4 years. 103. Miss R., 38.
Stage II.-III. in 4 years.
- $\frac{1}{2}$ year. 104. Mrs. B., 36.
Stage II.-III.—cough for 5 months; dead within a year.
- 2 years. 105. Mr. O.
Stage III. in 2 years.
- 1 $\frac{1}{2}$ years. 106. Mr. H.
Stage III. in 15 months "when baby was ten weeks old."
- 2 years. 107. Miss H., 20.
Stage II.-III. in 2 years.
- 4 $\frac{1}{2}$ years. 108. Mr. E.
Stage III.—"fistula" 4 $\frac{1}{2}$ years ago.
- 2 years. 109. Miss S., 25.
Stage II.-III. in 2 years.
- $\frac{1}{2}$ year. 110. Miss G., 18.
Stage II.-III. in 9 years.
- 1 year. 111. Miss W.
Stage III. in year or 1 $\frac{1}{2}$ years.
- 7 years. 112. S., 32.
Stage II.-III.—7 years.
- 3 years. 113. Miss M., 19.
Stage III.—acute pleurisy 3 years ago.
- 20 years. 114. C.
Stage II.-III.—20 yrs. ago pleurisy and large hæmorrhage.
- 1 $\frac{1}{2}$ years. 115. W. R.
Stage II.-III. in 18 months.
- $\frac{1}{2}$ year. 116. Mr. H.
Stage III. in 2 or 3 months.
- 1 $\frac{1}{2}$ years. 117. P. H.
Stage III.—1 $\frac{1}{2}$ years ago hæmorrhage.
- 2 years. 118. B.
Treated by Dr. C. R. for indigestion then sent to country.
9 st. 12 lbs. Yet Stage III. in 2 years.
- 1 year. 119. G., 22.
Stage III. in 1 year.

- 2 years. 120. Sister M. P.
Stage III. in 2 years.
- 2 years. 121. Sister M. A.
Stage III. in 2 years.
- $\frac{1}{2}$ year. 122. V.
Stage II.-III. in 7 or 8 months.
- $\frac{1}{2}$ year. 123. W. H.
Stage III. in 9 months.
- 3 years. 124. L.
Stage III. in 3 years.
- $\frac{1}{2}$ year. 125. A., 24.
Stage III. in about 3 months.
- 1 year. 126. F.
Stage III. in less than a year.
- 4 years. 127. M.
Stage III. in 4 years.
- $\frac{1}{2}$ year. 128. F., 36.
Stage III.—"six months ago quite well."
- $\frac{1}{2}$ year. 129. Miss R., 18.
Stage III.—cough for 5 months.
- 2 years. 130. B. G., 19.
Stage III.—2 years before Drs. H. and P. said nothing the matter.
- $\frac{1}{2}$ year. 131. J.
Stage III.—6 months ago quite well.
- 2 years. 132. C.
Stage III. Dr. K. found no trouble 2 years ago.
- 1 year. 133. M., 43.
Stage III. in a year.
- 3 years. 134. Miss C., 19.
Stage III. in 3 years, hemorrhage 2 years ago.
- 1 $\frac{1}{2}$ years. 135. S.
Stage III.—c. tuberculous laryngitis in 15 months; died in a year.
- 6 years. 136. Mr. C.
Stage III.—pleurisy 6 years ago.
- $\frac{1}{2}$ year. 137. Miss S.
Stage III.—8 months ago influenza, "went away and got better."
- 2 years. 138. Miss P.
Stage II.-III. in less than 2 years.

139. Miss B.
 1 year. Stage III.—"cough for six months."
140. Miss G.
 3 years. Stage III. in 3 years.
141. Mrs. P.
 1 year. Stage III.—"cough for a year."
142. Mrs. S.
 2 years. Stage III. in less than 2 years; in sanatorium for 9 months.
143. Mrs. S., jr.
 7 years. Stage III. in 7 years.
144. Miss MacP., 25.
 2 years. Stage III. in less than 2 years.
145. Mr. B., 22.
 1 year. Stage III.—"cough a year or more."
146. Mr. W., c. tuberculous laryngitis.
 1½ years. Stage III. Vet Dr. B., throat specialist, said "strained voice."
147. Mr. M.
 ½ year. Stage II.-III. with laryngeal tuberculosis; slight cough for 6 months.
148. A., 30.
 2 years. Stage III. in 2 years; died in 6 months.
149. W., 20.
 1½ years. Stage III.—17 months ago hemorrhage.
150. P., 52.
 1 year. Stage III.—cough for 12 months.
151. G. P., 22.
 1½ years. Stage III.—cough and cold for 15 months.
152. Mr. A., 29.
 ½ year. Stage III., and ulceration of tonsils and palate. Vet 6 months ago no cough or phlegm.
153. Mr. F.
 2 years. Stage II.-III. in 2 years.
154. N., 21.
 2½ years. Stage III.—c. pneumothorax; 2½ years ago hemorrhage aspirated in fluid five times by Mr. C. F. B. C.
155. Miss P., 27.
 1½ years. Stage III.—cough for 1½ years.
156. Mr. H., 22.
 1 year. Stage III.—pleurisy 12 months ago; died in another 6 months.

- 3 years. 157. W. T., 34. Febrile.
Stage III.—1 years ago cough.
- 10 years. 158. Mr. K., 53.
Stage II.—III. in 10 years.
- 1 year. 159. Miss S.
Stage II.—III.—"cough for 3 weeks."
- 30 years. 160. Mr. E., 53.
Stage III.—30 years ago blood-spitting.
- 2 years. 161. Miss B.
Stage II.—III.—"cough for 2 years."
- 2 years. 162. Miss D.
Stage II.—III. in 2 years.
- 2 years. 163. Miss M.
Stage III. in 2 years.
- 1 year. 164. Mrs. W.
Stage II.—III. within a year.
- 1½ years. 165. Mr. W.
Stage III. in 1½ years.
- 1 year. 166. Miss B., 17.
Stage III. within a year; came from New Zealand 3 months ago and gained 14 lbs.
- 1½ years. 167. H. J., 26.
Stage III.—cough 15 months ago.
- 2 years. 168. Mr. J., 35.
Stage III.—cough 2 years.
- 3 years. 169. T.
Stage III.—3 years ago pleurisy, and sent to country for 14 months.
- 1 year. 170. M., 34.
Stage III.—c. tuberculosis of larynx; cough for 6 months.
- 1½ years. 171. H.
Stage III.—1½ years ago quite well.
- 2 years. 172. Mrs. W., 21.
Stage III.—cough for 2 years.
- 1 year. 173. Miss C. B., 22.
Stage III.—cough 12 months ago, called "catarrh" by Dr. O. K.
- 2 years. 174. J.
Stage III. Did not know he had phthisis till he came to me; under doctor for 5 or 6 years.
- 1½ years. 175. W., 53.
Stage III.—"cough 16 months ago"

- 1 year. 176. Mrs. S., 43.
Stage III.—c. tuberculous laryngitis, "cough 12 months."
- 1 year. 177. Miss T., 22.
Stage III. A year before Dr. L. more afraid of asthma than consumption.
- 2 years. 178. M. T., 45.
Stage III.—cough 2 years ago.
- 1 year. 179. M., 43.
Stage III. in a year.
180. P.
1½ years. Stage III.—c. tuberculous laryngitis. Dr. S. J. said 1½ years ago nothing the matter with lungs.
- 1 year. 181. Miss C., 37.
Stage II-III. A year ago Dr. B. said lungs were sound.
- 6 years. 182. M. F., 37.
Stage III.—cough for 6 years; 9 months in sanatorium, where he had severe hemorrhage (7 pints).
- 1 year. 183. W. S.
Stage III.—hoarse for 3 months; cold a year ago.

CHAPTER IV

GENERAL TREATMENT

(A) *Climate as a Factor, with special reference to the Climate of New South Wales.*

THE problem of selecting a suitable climate for various cases of pulmonary tuberculosis is constantly presenting itself to us in our daily practice, and its proper solution may after all defy all the inquiry and thought and discrimination that we bestow upon it. Each one has, in a sense, to work out the problem for himself, and as the x , y , z 's have a varying value, exact comparisons are hardly possible. My own conclusions are so completely at variance with the current opinions of the hour—opinions firmly rooted in the minds of some of our leading practitioners, and more or less clearly reflected in the mind of the laity—that the time has come for me to give reasons for the faith that is in me; if so be we may thus at least gain a clearer insight into the complexity of the problem and realise that the vision of truth is still very far off.

In Europe, climate as a factor in the treatment of disease has been studied on more or less haphazard lines in the numerous sanatoria, watering-places, and institutions that are scattered in all directions, but even at these places vested interests may militate against the discovery of truth; yet gradually, with the progress of time and knowledge, special climates have, by implication, been stripped of the great virtue that was once thought to be their exclusive possession. Accessory conditions, such as fresh air, sunshine, careful diet, graduated exercises, massage, and the continued use of mild drugs acting on different organs, are now claimed to possess the royal virtue originally credited to climate.

In Europe one observes a marked change of opinion as to the value of climate in the treatment of pulmonary tuberculosis. In Australia we have not even the experiences of sanatoria to help us. In spite of the existence of Mr. Goodlet's noble work at Thirlmere, a sanatorium at Echunga and at Mount Macedon, and one at Roma, we have very little positive relevant evidence on which we dare express any but a haphazard opinion as to the value of climate in the treatment of pulmonary tuberculosis. The cautious will therefore hold their opinions in reserve, and shun that dangerous form of dogmatism that is often rampant when truth is entirely beyond our reach, and logical methods are not equal to the task of proving our errors. On the nowadays all-important problem of the relation of climate of any or every sort to the treatment of pulmonary tuberculosis in the early stages, dogmatism cannot help us, and will probably lead us astray. As yet it is hardly possible to define precisely what we mean by the early stage of lung tuberculosis. I should like to restrict this term to the stage of the disease before tubercle bacilli are present in the sputum—when the tuberculous lesion is still closed or shut off from the external world, represented by the air passages. This definition would have this advantage, that medical men and the laity alike would understand the form of tuberculosis under discussion, and would also have it firmly impressed on their minds that the disease may have possession of its victims months, and even years, before the obvious symptoms of the disease develop. At present many medical men and the laity consider that pulmonary tuberculosis of necessity causes fever, cough, and wasting. On the contrary, it is true that tuberculous disease of the lung may never show its existence by any symptoms, as *post-mortem* examinations continually testify, and in other cases may exist for a very long time without even attracting the attention of patient or doctor. It is not impossible that medical men often raise groundless fears about weak lungs, and on the other hand overlook the existence of disease that can be recognised by proper methods. If tubercle bacilli are found in the sputum, the stage is relatively late and the golden opportunity for treatment in the

early stage may be lost. In this early stage there may be no fever, no expectoration, even no cough. I have seen a case of closed tuberculosis involving a large part of the upper lobe of the right lung in a man who for years searched the globe in vain for the climate that would make him well. Physical signs were altogether out of proportion to his local symptoms. On the other hand there may be but slight physical signs till a hemorrhage unexpectedly occurs, and too often the first stage rapidly passes into the second stage, usually with the signs and symptoms of secondary infection. Such cases are totally unlike in every respect, and yet they are both cases of closed tuberculosis till the hemorrhage blots and blurs, and may ruin for ever, the pure type of the process. Otherwise such cases are cases of pure tuberculosis. Again, in tuberculosis involving the pleura, the first positive evidence of the disease may be acute pleurisy suddenly attacking a person apparently in sound health, and soon developing into pleurisy with rapid effusion and all the accompanying trouble and danger; or, on the other hand, there may be extensive disease of the pleura without any acute process, but ending in adhesions and thickening, sometimes running a latent course for a long time. This state, then, in which expectoration hardly occurs—and tubercle bacilli are absent from the expectoration—is the basis of classification for many, especially in the most modern sanatoria for the cure of tuberculosis among the poorer classes. If this stage is past the case is rejected (Weicker). As soon as tubercle bacilli enter the air passages and escape in the expectoration, there must be destruction and disintegration of the lung tissue at the surface, and then the risk of secondary infection is imminent. Till then the process is tuberculosis pure and simple. Then it is that the flood-gates are open, and organisms of various kinds, streptococci and diplococci mainly, may find a suitable nidus for growth and development in this disintegrated tissue, whence the tubercle bacilli escape. As tubercle bacilli leave, the organisms of septic processes enter by the door, and the whole character of the process is profoundly changed. No doubt this secondary infection that brings the process to the second stage

may be long delayed, especially in pure air, by conditions that we little understand, but once it is established fever shows itself, and on the chart this infection asserts its origin by the zigzag course of the temperature curve. Anæmia, wasting, sweating, diarrhoea, and other symptoms may in the course of a few weeks demonstrate the severity of a twofold toxæmia. There may even be a degree of septicæmia. But even at this stage the case may not be hopeless. Change to a pure air in the mountains or plains—or at the seaside—with other measures, may get rid of the secondary infection, and the tuberculosis making a pretence of slumbering for a while, may progress so slowly that it causes hardly any symptoms. The expectoration diminishes—even tubercle bacilli may disappear, and in favourable cases the lung heals. Thus the disease may pass from the second to the first stage. More often the expectoration is scanty, and some few tubercle bacilli can be found. The disease may seem to be in the first stage though it is in a latent second stage—ready at any moment to become aggravated. It may require all the bacteriological skill of the physician to recognise this latest second stage. Often this *latent second stage* is nothing else than the passive secondary infection of other authors. Even after considerable destruction of lung tissue with the formation of irregular cavities the process may regress, and in rare cases end in healing of the ulcerated surfaces. Again, the process may return to the phase either of a latent second stage, or even of closed tuberculosis. For many, then, this state is the criterion. When the tuberculosis is thus closed, the first stage of the disease exists. I am convinced that if we can only provide a sanatorium for the treatment of these cases by the most modern methods¹ the public will have no reason to withhold its support from the philanthropic scheme that has been inaugurated for the treatment of tuberculosis among the poorer classes. I hope that I have given some precision to the otherwise loose and indefinite condition known as the early stage of pulmonary tuberculosis.

If my definition is accepted, I may state at once that we have no evidence for or against the value of this or that

¹ *Tubercula.*

climate in pulmonary tuberculosis in the early stages. This basis of classification has not existed, and it is idle to suppose that any physicians in Sydney or Australia have investigated the effect of different climate on this stage of tuberculosis. But even interpreting the early stage of pulmonary tuberculosis in the ordinary vague and varying sense, we can hardly get nearer the truth, for no physicians in Australia have attempted to discriminate between the effect of climate on these cases as distinct from other cases. At present we are not in a position to form any opinion of our own, much less is there any ground for dogmatism. Most physicians diagnose tuberculosis of lungs by fever. That symptom alone, if of moderate severity, is generally sufficient to show that the case does not belong to the early stage of simple tuberculosis. In judging of the effect of climate upon the course of *pure tuberculosis of the lungs*, in other words, of pulmonary tuberculosis in the early stages we must carefully put out of view the effect that is produced by climate, mountain or other, upon the second stage of tuberculosis, when secondary infection, with fever, is the dominant and grave condition. Any line of reasoning I use, leaves out of consideration the disease in the second stage when secondary infection is so common. On this phase of the question I have already explained myself. (See *Australasian Medical Gazette*, August 20th, 1897). How then are we to form an opinion concerning the effect of any climate upon the first stage of tuberculosis of the lungs? As we have no trustworthy evidence of our own, we may learn something from other sources, and especially from the views of recognised authorities. Thus we may be in a position to form some general conclusions that will guide us in condemning or favouring the sea coast of New South Wales in dealing with the early stages of tuberculosis of the lungs. It is well to say at the outset that we have no evidence to show how the climate of the sea coast affects early tuberculous disease of the lungs. Those of the older physicians who may be inclined to dispute this assertion hardly resort to the systematic examination of sputum for tubercle bacilli in their everyday practice. If they use this method, they use it when

it is hardly necessary. *Negative evidence*, unless obtained by the methods of the skilled bacteriologist, has no great value. Tuberculin as a diagnostic agent is not used at all, and the physicians, who cling to the old views, have neither the experience nor the knowledge necessary to determine the diagnosis of secondary infection. But my main objection is of a more serious kind. Whatever may be said to the contrary, it is certain that no observations have ever been made discriminating between the effects of the climate of the sea coast and the effect of the density of population in Sydney and Newcastle. The physicians in Sydney have attributed to the climate of Sydney and the coast effects that distinguish, and are a necessary accompaniment of all crowded populations irrespective of site or climate. It needs no argument to prove that Sydney, as a dense centre of population, will not show as good a record as other districts with sparse and scattered populations, but the effect is not at all the effect of climate, good or bad, but the effect of that crowding of masses and accompanying conditions of life that militate against health and specially favour the spread of infectious diseases, even of tuberculosis of the lungs. A fairer comparison may be drawn between Sydney and cities in the world of similar population. Such a comparison shows beyond doubt that so far as the incidence of tuberculosis of the lungs in the community is concerned, Sydney is one of the most favoured cities in the world, the incidence of tuberculosis of the lungs being less than 1 per 1000, according to latest returns. It is at least a fair argument to use that the relatively low incidence of tuberculosis in Sydney must be due to some special favouring local conditions, and one might even state that the climate of the sea coast, mild and equable throughout the year, is a factor in this relatively fortunate result. But soil and situation are factors. Until it is proved to be otherwise, I see no other logical conclusion than that, were it not for the density of the population, Port Jackson itself would form an admirable site for a sanatorium. And this conclusion is strengthened when we examine the incidence of tuberculosis in districts that possess a climate like that of Sydney, but lack the crowded population. The

North Coast and South Coast districts will serve for such comparisons. These districts contain many small scattered towns, such as Grafton, Lismore, Kiama, Wollongong, Nowra, and others, and have an aggregate population of about 140,000. They show an incidence of one death from tuberculosis in every 2,730 living persons. New England, with a population of 53,300, shows an incidence of one death from tuberculosis in every 3,520; Namoi and Gwydir, one per 2,360; Young and Gundagai, one per 2,470; Bathurst, one per 2,180; Hunter, one per 1,830; Mudgee and Argyle, one per 1,800; Murrumbidgee, one per 1,500; Murray, one per 1,490; Western districts, including Broken Hill, one per 470. In England, which is *facile princeps* in the application of hygiene to the needs of the nation, and enjoys also the honour of presenting the lowest death-rate from tuberculosis among all European nations, in spite of the fact that it is sea-bound and has no mountains worthy of the name, in spite of its rain and wind and fog, its want of sunshine and its uncertain climate, in England the death-rate from tuberculosis is one per 737 living persons, in United States (28 larger towns) one per 400 living persons, in New South Wales, one in 1,200.

At the great German Congress on Tuberculosis it was stated that Buenos Ayres showed the lowest death-rate of all the large states of civilisation—1.62 per 1,000 inhabitants—one death per 630 inhabitants. Sydney is better than Buenos Ayres—one per 1,080 (1900)—and, therefore, in regard to its mortality from tuberculosis shows the lowest record of all large cities. The learned German knew nothing of Sydney's fortunate best on record. Buenos Ayres, with a population of 567,342, lost 1,876 from pulmonary tuberculosis in the two years 1892-3. The metropolis of Sydney in 1899-1900, with a population of 480,000, lost 916. Roughly speaking, there are three deaths in Buenos Ayres from tuberculosis of the lungs for every two deaths in Sydney from the same cause. Sydney alone of all the great cities shows a rate of less than one per 1,000. It is worth while mentioning too that, comparing the largest cities in the world in which population exceeds 500,000, the towns on the seaboard show the lowest mortality from tuberculosis, Buenos Ayres, Naples, Amsterdam, London, and Hamburg.

Accordingly sea air in the northern hemisphere—even in the warm Mediterranean—in no way favours the tuberculosis process. Even in the southern hemisphere Buenos Ayres is no exception. If, therefore, it is asserted that that sea air in our region of the southern hemisphere brings disaster to cases of tuberculosis, we may surely ask for something more than assertion—we want proof. So far as I am able to sift evidence the proof is quite the other way. A simple analysis of the death-rate from tuberculosis in the districts of Sydney, which I take from the valuable and laborious commentary of Dr. W. G. Armstrong, Medical Officer of Health, confirms my views in the most startling manner. The combined districts of Vaucluse, Mosman, Hunter's Hill, and Lane Cove, with a total population of 10,575, had not one single death from tuberculosis to its account in 1899—a very remarkable record, showing at least that the sea air on the coast does not favour tuberculosis. Randwick, with a population of 8,225, shows only a rate of 0.24 per 1,000 in 1898, i.e. less than 1 in 4,000, a far more favourable rate than exists in any country district. Manly also has a low rate.

If, then, we compare, not the districts of the wealthy, but the districts of the poor, we find that Balmain last year, with a population of 28,675 distributed with a density of 32 to the acre, has only a rate of 1.05 per 1,000. This favourable rate is a strange commentary upon the present view of the profession with regard to the injurious effects of the sea air and sea coast upon tuberculosis of the lungs.

Let us now turn to the coastal districts of New South Wales. In what way a climate, that presents a low general mortality, affects tuberculosis of the lungs it is difficult to say, but probably, if a climate is very favourable to general health, it will not be unfavourable to those suffering from early tuberculosis. The North and South Coasts present incomparably the lowest death-rates of all the districts in New South Wales—North, per 1,000 in 1897-9—7.1, 6.9, 7.9. South Coast, 6.8, 6.8, 7.7. No other districts approach these districts in the low death-rate. New England comes next with a rate of 8.2, 9.8, 10.1 per 1,000.

There is another thought worthy of attention. If there is one firmly-rooted conviction in the minds of all who have considered the question it is that predisposition plays an important part in the origin of pulmonary tuberculosis. Some exaggerate the importance of this factor, hardly anyone refuses to recognise it. We can hardly explain the nature of this disposition of tissue to invasion by the tubercle bacillus, nor have we a right to say that this or that nation exhibits a greater disposition to the disease. Yet, if we hold that predisposition shows itself in families or groups, we should be able to apply this view on a larger scale to nations. In such a case we might well argue that the British race exhibits less predisposition than any other race, because the British nation suffers less than any other European nation. The French nation suffers twice as much from tuberculosis, and Russia three times as much as Great Britain, at any rate in the large centres of population. As soon as we begin to argue that other conditions account for this difference we are at once knocking the bottom out of the view of predisposition. If, however, we accept the doctrine of predisposition as the first factor of importance, as some still maintain, our nation—the grand old British race—shows collectively a far lower degree of predisposition than other nations. Switzerland with its mountains and their vaunted power against tuberculosis shows an incidence of tuberculosis in the population of 2,031 per 1,000,000; England, 1,358 per 1,000,000; New South Wales shows an incidence of 806 per 1,000,000 souls. The conditions in New South Wales modify the incidence of disease among Englishmen and their descendants. In New South Wales every year there are 500 fewer deaths per 1,000,000 from tuberculosis than there are in England. Now if predisposition means anything it means some definite and permanent character of tissue. It can have no particular meaning if it can disappear in a generation. Experience seems to tell us that the disposition to tuberculosis is consistent with robust health and fine physical development. We are therefore not justified in believing that the conditions in New South Wales suddenly modify the predisposition of Europeans to tuberculosis. For no reason at all the idea

gained currency that the Australian-born were more liable to tuberculosis than their English-born parents. This is just a fair example of the readiness with which people indulge in reckless theories that rest on no foundation at all either of observation or fact, and may live as long as careful observations on the point at issue are wanting. There is hardly any more substantial basis for the view that the sea coast of New South Wales is not suited for cases of pulmonary tuberculosis. On the other hand we must not flatter ourselves with the notion that the mortality statistics from tuberculosis in New South Wales are vitiated by the intrusion of a number of imported cases of tuberculosis. In 1899 there were 1,069 deaths from tuberculosis. Of these no fewer than 600 were born in Australasia; 220 more had lived in Australasia twenty years or more; 165 more had resided in Australia ten to fifteen years; while only 58 had been less than five years in Australia. We are justified in saying that 90 per cent. of the cases had their origin in Australia, while 60 per cent. were among the native born. It would require a careful analysis of living persons who are native born and importations to show whether the native born show a greater or less disposition than the European. Now, if the predisposition exists in the same degree in the British race, whether imported or native born, we must hold that there is still a large contingent of persons prone to the disease. The disease has not yet attacked the full complement of predisposed. The soil is ready, but the seed has not been sown. In our community above all others, every effort should be made to prevent the disease extending its ravages to the full complement of predisposed. In short, prophylaxis will be of even greater benefit in Australia than in the older countries. Yet we are behind the world in measures of prophylaxis. There is not even a general hospital for consumption, which educated public opinion should urge the Government of the State¹ to establish, in order to diminish in some measure the numerous foci of infection that are a source of danger to those who live on terms of close intimacy with those suffering from the disease.

¹ 1907. At last Government has awakened to the necessity for such institutions.

It is interesting also to observe the mortality rates in Victoria and Melbourne respectively, and in Queensland and Brisbane and compare them with those of our own State. In 1899 the death-rate in Queensland was 12.07; in Victoria 14.28; in New South Wales, 11.82. In Melbourne, with a population of 470,000, the rate of mortality from tuberculosis of the lungs has varied from 948 to 654, the average being above 700. In Sydney, with the same population, the annual death-rate from the same cause is 481 (1899). This excludes ninety cases that died in the Liverpool Asylum, of which the majority belong to the metropolis. Even allowing the rate in Sydney thus corrected to be 360, we find a difference greatly in favour of Sydney. The conditions of life in Melbourne kill, by tuberculosis, 180 to 200 more persons every year than the condition of life in Sydney. Is this an argument for protection? The same advantage is evident also in the conditions of life in the colony generally. Victoria loses every year from tuberculosis, 300 to 600 more than New South Wales, even though the population of New South Wales is greater. In Queensland and New South Wales the death-rate from tuberculosis is about the same, while Brisbane with its smaller and far more scattered population, shows a better record than Sydney, 1 in 1,450. Indeed relative to the population, the death-rate is better than that of the colony taken as a whole; if indeed the area of the city is the same for the collecting of mortality and population statistics. There may be some local conditions, such as the situation of institutions outside the city area, that would place these deductions from statistics in a different light. If, however, these statistics are correct, *Brisbane has an enviable position with regard to tuberculosis*. I know no other country in which the rate of deaths from tuberculosis is relatively less in its capital than in the remainder of its population from the same cause. So far as these figures serve us, we find that a low general mortality means a low mortality from tuberculosis; indeed, that the mortality from tuberculosis is an index of the general health of the community, and conversely we may say that when the general death-rate is low, then the rate from tuberculosis will be low. No country in the world

can show a lower death-rate from tuberculosis than New South Wales, and in New South Wales the coastal districts show a better record in this respect than any other district with the single exception of the New England district. *Even the New England district shows a rather worse record than the South Coast in respect to the incidence of tuberculosis of all forms.* But no other districts approach these districts in their relative immunity from tubercular disease. The coastal districts further show a far lower death-rate from pneumonia, bronchitis, and other diseases of the lungs. In 1899 there were ninety-six deaths from pneumonia and bronchitis in New England (population 63,290), but only thirty-four deaths in the South Coast (population 62,740), and forty-nine deaths in the North Coast (population 78,300). Diseases of the lungs are a good index of the variability of the climate. In no other district is the incidence of diseases of the lungs so small as in the coastal districts. These statistics undoubtedly seem to establish the fact that the climate of the North and South coasts is unusually propitious, and inasmuch as the incidence of tuberculosis in these districts is lower than in any other district I personally shall require further evidence before I give up the attitude I have long adopted in recommending strongly the coastal districts of New South Wales for the treatment of tuberculosis.

I wish, therefore, to put forward the following proposition: "That the air of the sea coast of New South Wales, far from being injurious to those suffering from pulmonary tuberculosis in the early stages, is beneficial, and may have as much curative virtue as any other climate in the world." This proposition is well worthy of discussion and of a collective investigation by the local branch of the British Medical Association.

I have shown that in this coastal climate there are fewer deaths from tuberculosis per 1,000 than in any other part of New South Wales. In 1897 there were only twelve deaths from tuberculosis in the population of 78,300 in the North Coast district. The conditions, therefore, cannot favour the disease nor hasten its course. The mild equable climate, free from great variations in temperature, and with a cool, refreshing north-east wind from the sea supplying relatively germ-free

air during the hotter months, from January to April, and during the cooler months with winds that are less severe and more propitious than the cold, piercing winds of the mountains, with a mean summer temperature of 70° to 75° F., a mean winter temperature of 53·5° F., and a mean annual temperature of 63·2° F.—such meteorological conditions would *a priori* lead one to anticipate a favourable effect, while experience tells us in plain and unmistakable language that these conditions cannot be bettered anywhere in Australia, if in the world. No doubt we must be ready to meet and dispose of special pleading. My views are heretical, and, as happened in the days of the Inquisition and Galileo, such views can only win the day on the transparent basis of truth. I court inquiry; I seek for full investigation. Even if my views should prove in the end to be exaggerated or unsound, the investigation will give us a more excellent view of truth. I have no other interest in this matter than to seek and find the truth. *Prima facie*, my views, supported by the evidence of statistics, bear the semblance of truth. Until numerous facts and observations are collated and given their proper place and order, truth cannot be established. At present we have no evidence to the contrary that is worthy of the name, no evidence that would pass muster in a court of law, or even of reason, and I throw down the gauntlet as the champion of the new view that the sea coast of New South Wales is not unsuitable for the treatment of pulmonary tuberculosis in the early stages or even in the later stages. I have suggested that the low mortality from tuberculosis in Sydney and the coastal districts is evidence in itself that the conditions obtaining in these parts are of advantage in the treatment of tuberculosis, and should be exploited. There are those who even now hold the opinion that the low rate of mortality from tuberculosis in Sydney and the coastal districts *is*—not may be—in part the effect of medical and lay opinion in condemning these districts. As a consequence of this condemnation, so these special pleaders argue, those who suffer from tuberculosis in Sydney and near the coast pack up their portmanteaux and hie to the mountain districts or the plains, where they die. Thus Sydney presents a more

favourable record, and the districts of immigration present a less favourable record than the conditions of the respective areas would justify. Such emigration and immigration, however, should have the result of showing a greater number of deaths from tuberculosis in the country districts than would be found in other places and other countries of similar populations. On the contrary we find that the death-rate from tuberculosis in the country districts of New South Wales is not only not high but exceptionally low. I have also the support of Dr. W. G. Armstrong in saying that so far from the death-rate in the country districts being increased by this exodus of tuberculous cases into the country, there is on the contrary an exodus from the country districts into the town. Dr. Armstrong writes: "Very many persons who died in Sydney hospitals came from country districts. Some few deaths of Sydney residents have occurred in country hospitals, but," he adds, "the number of deaths occurring in the country which ought to be credited to the metropolis is, however, certainly insignificant." This may or may not apply to tuberculosis, as the statistics are purely the statistics of the Sydney hospitals. One may say that many go, or are sent to the country in the earlier stages, but few remain there to die. They seek the country, too often in vain, for a cure, but that failing, they return home to die. Certainly my own experience tells me that many of the worst cases I have seen have lived all their lives in the districts with vaunted climates. From the west I have had many cases; from the North Coast, none; and but a single case from the South Coast. Now it is to work out accurately the problem in this direction that we should have a collective investigation. The investigation could be easily carried out by drawing up forms to be filled up by medical men in the various districts. The forms should be drawn up in such a way that the country practitioners should be able to state the number of cases that have *died* under their care, the birth-place, length of residence in the district, and such other information as will show the incidence of the disease among those who belong to the district and those who have come from other parts, especially from Sydney and other coastal

towns or districts. It is very remarkable that the districts in the immediate neighbourhood of Sydney should present such favourable records. One would expect that residents in these districts would be more often exposed to the risk of infection by the mere proximity which facilitates intercourse with the population of the dusty and crowded city. Yet in one year the North Coast, including Grafton, Lismore, Ballina, Maclean, Bellingen, Port Macquarie, Kempsey, Casino, Taree, and other places, had but twelve deaths from tuberculosis in the whole district. There is at present not much to support such special pleading. However, until we have the results of a collective investigation such as I propose, I am absolutely justified in holding the opinion that the district of the sea coast is as suitable as any other for the treatment of pulmonary tuberculosis. A sanatorium for the poorer classes should be in a situation within reasonable access of Sydney for many reasons. It can be built and maintained at the lowest possible cost. It enables the friends and relations of the inmates to visit the sanatorium without much cost. This will minimise the sense of isolation that may otherwise be a bar to the prolonged stay in the institution that is indispensable for satisfactory results. A happy state of mind contributes to the success. Most important of all, there can be thorough supervision, even from Sydney. Of course a resident medical officer is indispensable, but I should hope that as in Vienna and elsewhere a central committee of medical men, of special knowledge and experience, should control and guide the principles of treatment in the institution. These are immense advantages that cannot be lightly set aside, because certain individuals, medical and lay, have the notion, formed from their own personal experience or from a limited and indiscriminate experience, that the mountain air has specific virtues. I grant that there is abundant testimony from the older physicians in favour of treatment at higher altitudes. But the pendulum of opinion swings now in this now in that direction. Not very long ago the older physicians firmly believed and taught that consumption was more easily cured in a warm climate than in a cold one. There was no foundation for this view, and accordingly cold climates in time came into fashion. Patients were ordered

to go to the Andes instead of Madeira or Egypt. Yet Koch tells us that tuberculosis plays no part at all in the mortality among inhabitants on the east coast of Africa—in the heat of the tropics where malaria reigns supreme. The great pioneer of the rational method, Dr. Brehmer, of Goerbersdorf, strongly advocates mountain air on account of the diminished atmospheric pressure, of the lower degree of moisture, of the great warmth of the sun, of the intense sunlight, of the rarity of fogs, and the purity of the air, especially its freedom from microbes on account of the sunlight. Sir Herman Weber admits that it is difficult to prove therapeutic results by statistics. He has seen 36 out of 144 cured—a misleading term—by mountain air. In 52 of the cases no improvement or downward progress to death. Better reports have appeared, especially those of Dr. Turban, but Dr. Turban and others have also adopted the rational method, based on dietetics, physical exercise, and plenty of fresh, pure air. Dr. Turban, too, has spoken favourably of tuberculin treatment in his institution. No authority of any eminence claiming the specific virtue of mountain air, has ever attempted to differentiate between the effect of the mountain air and the effect of the numerous measures that constitute the rational system of Dettweiler, Dr. Brehmer's pupil and successor. Sir Herman Weber, who is the special advocate for mountain air, says that in cases of circumscribed lesions almost all climates can be exploited with good effect, though (he adds) in robust constitutions especially mountain climates and sea voyages. After pointing out indications for special climates, in which one observes that he highly commends warm climates with plenty of sunlight, he adds: "The cure of tuberculosis in the early stages is possible in all healthy climates." Listen to this peroration: "But climate alone, without careful medical supervision, is for the most part unsatisfactory. The blind faith of patients in climate leads often to failure, to progress of the disease and death. Therefore, for the majority of patients, treatment in sanatoria is to be preferred, but for the poorer classes is a necessity. The erection of sanatoria for the people is a national necessity for the cure, the prevention, and the eradication of tuberculosis."

Even Sir Herman Weber places greater virtue in the methods of the sanatorium than in any climate.

In the latest text-book on medicine we read: "It is only during the winter, however, that the climate of Davos or of the Engadine has special advantages." In the still dry air patients can bear the great cold. Appetite is stimulated, and much food can be taken. The climate of Noodrach is by no means a climate that has any special advantages, if we accept the account of a physician who was cured at this famous sanatorium.

Dettweiler, disciple of Brehmer, is the great apostle of sanatorium treatment on hygienic, dietetic, and physical principles. The disciples of this, the latest school, in the treatment of consumption, set but little store on the effect of climate, whether of mountain, sea, or plain. To use Dettweiler's own words, "This new method consists in curing the disease by the method of rest and open air carried out in its smallest details according to the strength of the patient and the stage of the disease, even during the night." This system renders it possible to treat consumption in all climates free from extremes and even allows patients to remain at home. In detail, it consists of hardening the skin by proper use of air, light, water (baths, douches), moist and dry friction, massage, in gymnastic exercises of the chest and whole body, by climbing, etc., in a systematic application of all physical remedies, in exercise or the reverse, according to the state of the patient—a system gradually and carefully adapted to circumstances in a manner that only a special institution can apply and control. This constitutes sanatorium treatment on a rational basis, that claims superiority over all other methods. Thus, "the disease can be healed in all good climates with equally good results." This is the open-air treatment. It would not be right to call it open-air treatment if certain climates were necessary. Even some of our physicians loudly talk of open-air treatment and recommend it, and yet cling to the older view that the air of the mountain is necessary. Worse still, they argue that the climate of our coast is not suitable for this method. Those who preach this gospel have but imperfectly understood the

new teaching. If one advocates the open-air treatment, one must follow those whose experience has demonstrated its value, independent altogether of special climates. We entirely misunderstand the experience and teaching of the new school if we say that a sanatorium for the people should be erected in the mountains rather than near the coast. In my limited experience I should hesitate to send patients to our mountains, with their variable climate, their mists, and their cold winds. A few robust men and women might do well. I doubt if the mountain climate would suit the majority as well as the mild climate of the coast. Turban, whose sanatorium is at Davos, writes that Dettweiler has especially accomplished the great task of demonstrating that the carrying out of the open-air method is not bound up in any special climate. Even the increase of the red corpuscles and of the hæmoglobin value of the blood—with increase of volume, such as may be the result of high altitudes, is after all a compensating process with a physiological explanation. The most modern sanatoria have not been erected at high altitudes. Falkenstein, Hohenhonnef, Nondach, Alland, near Vienna, the sanatoria near London, Edinburgh, and Liverpool, in effect disregard the value of mountain air. Dr. Sydney Jones has himself mentioned statistics from open-air treatment near London, which we may strive to imitate but can never excel. Dr. Sydney Jones stated that *75 per cent. had been cured*. They will never record such statistics again. Yet the climate of the sea coast of New South Wales is as favourable as that of London: I should say far more favourable. I could quote authority after authority, emphasising the great value of sanatorium treatment, and by implication paying little attention to mountain air as an appreciable factor in the new treatment of pulmonary tuberculosis. The new treatment consists essentially in open air, dietetic regulations, and physical training. Of quite subsidiary importance is the air of the mountain, of the sea, or of the plain.

I might even, with reason, venture to discuss a second proposition, viz., that the climate of the mountains near Sydney, Katoomba, Mount Victoria, etc., is not as suitable as the climate of the coast for sanatorium treatment, but I wish

chiefly to test the truth of the firmly rooted opinion that the climate of the sea coast is not suitable. If we arrive at the conclusion that this climate is eminently suitable for a sanatorium for consumption, there are very great obvious disadvantages in erecting a sanatorium on the mountains. The want of a good water supply, the difficulty if not impossibility of keeping cows for the all-important milk supply throughout the year, the difficulty of growing vegetables and fruit trees, which would in other places offer good and pleasant occupation to the inmates of the institution, are among the most obvious disadvantages. Hence the selection of a site in the mountains can only be justified when it can be shown that these disadvantages are more than counterbalanced by the virtues that have not yet been proved to exist in the air of the Blue Mountains.

Long-established error is often a vigorous weed that blights every healthy growth, and can only be uprooted by dint of much labour and patience. If error is closely intertwined with vested interests or personal reputations, it may be difficult to kill the parasite, but the demands of truth and the public interest far transcend in importance either vested interests or personal reputations. Otherwise, the fallacy, if, indeed, it be such, that now possesses the public mind, might well be left to die without a murmur—without a regret. If my views should ultimately win the day on the eternal basis of truth, the public gain will be great. Many an unfortunate victim may still have to bear his disease, but he need not suffer exile in strange and distant places, where he is bereft of the pleasures and comforts of society, even of the necessities of a healthy and happy life. Surely, in the interests of the whole community, we should lose no time and spare no energy in discovering by a collective investigation, whether I am right or wrong in saying that neither experience nor facts justify the profession and laity in perpetuating a gross libel upon the beautiful and health-giving climate of the sunny coast of New South Wales.

1908. *The Aftermath of this Article, written in 1901.*

The preceding article was published in 1901 and aroused a violent storm of indignation and opposition. The leader of this opposition promised to deal with the subject at length in order to show that my statistics were faulty and my conclusions utterly unsound. I have waited in vain for seven long years for a rebuttal of my evidence. In those seven years the majority of the profession have been converted to my views, and many doctors, who used to send their patients to the mountains and into the country, now send them to the district near Sydney where I established my own private sanatorium. Moreover at this sanatorium I have had results which place my original contention beyond any doubt. In 1905 I had forty-five cases of pulmonary tuberculosis under treatment in this climate from November, 1904, to April, 1905. I specially selected the very worst months of the year for my observations. During these months the atmosphere is apt to be close and enervating, and sudden changes of temperature are frequent; favouring chills, and depressing vitality. Of the forty-five cases under treatment, three lost a few pounds. One of these was in the third stage, but it was a loss of but 3 lbs. The other two, who lost weight, lost their disease also—to all appearance—so that no great harm was done. In the remainder—forty-two cases in all—there was an average gain in weight of *eight pounds*. This extraordinary gain in weight in spite of disease at a time of year when most people lose both weight and energy in a climate heartily condemned by the medical profession of Sydney, was no doubt largely due to tuberculin, but at least we may say that the conditions of climate were not adverse. However, if the medical profession admit that tuberculin was the effective cause of the great improvement, I am quite content. If they have been converted to my views regarding the healing virtues of tuberculin they will in time be converted to my views upon the health-giving virtues of the sunny coast of New South Wales.

(B) Treatment by means of Sanatoria without Tuberculin.

Nowadays the air rings with the praise of the sanatorium in the treatment of pulmonary tuberculosis. The great German physician Brehmer suffered from consumption, and cured himself at Goerbersdorf. He introduced a new system without entirely breaking away from the teachings of Hippocrates, Galen, and Celsus. He wrote in 1854, "Pulmonary consumption is curable; it is curable under suitable climatic conditions (mountain air) by a hygienic-dietetic system of treatment carried out in an institution." His new system was ridiculed as a fad, but his masterful energy and supreme confidence won slowly, and in spite of much opposition, the consideration and approbation of all. His chief disciple, Dettweiler, elaborated the system of Brehmer, and added to it "Rest" as an essential factor. Thus rest, abundance of pure fresh air, energetic feeding, strict management of the daily life of the patient under the watchful eye of a specialist, hardening of the system by living in the open air, and hydropathy constitute the essentials of Brehmer's system.

The theories upon which Brehmer based his methods are forgotten, but his practice showed that pulmonary tuberculosis might be cured, not by climate *per se*, but by sanatorium methods carried out in any good climate free from extremes. Many a consumptive may therefore be treated with success in his own home. It is equally certain that this method of treatment cannot be applied to the poor in their own homes. The poor therefore cannot be treated except at the sanatorium. Moreover, it is daily becoming apparent that a residence of at least six months in the sanatorium is necessary. Thus the system is in its essence costly and therefore beyond the reach of the majority of the poor. If we consider the prevalence of the disease among the poor, none but a visionary can suppose that such a costly method can help the majority. As a means of bringing respite, and occasionally cure, to those who have money and time at their disposal, the system may commend itself, but I am persuaded that hundreds and thousands have learnt to their disappointment and sorrow that even under favourable circumstances the sanatorium has

its conditions and limitations. Sanatorium methods generally effect immediate improvement, and if the patient can spend some months each year under these special conditions, his life will be prolonged by some years, and in some cases the disease will be even completely cured.

Sanatorium treatment is not open-air treatment. Sanatorium treatment is certainly not the treatment recommended by the mass of medical men in Sydney. There is a strange, unreasonable and unwarrantable prejudice against the sea air of New South Wales, which leads every doctor in Sydney to think that he has solved the problem of appropriate treatment for consumptives if he despatches them into the country to get away from the sea air. I have already dealt with this current prejudice, and have but to add that, in my own experience, I have found the air of the sea coast of New South Wales to be no unfavourable factor in the treatment of consumption. This haphazard system of sending into exile all who suffer from consumption is not sanatorium treatment. Sanatorium methods can be carried out quite as well within twenty miles of Sydney as in the Blue Mountains, or at Hay or Wellington or Orange or Bathurst or Armidale. I have had very serious cases of consumption occurring among the native born of these localities, and if the climate will not prevent the disease, it is not likely to be a powerful factor in curing it. (See Appendix.) Medical men in Sydney often pack off their patients to the country without any instructions. They do not even tell them to take their temperature lest they should get too fond of taking it, as if it were some fatal, fascinating beverage. The importance of "rest" is ignored. They are merely told to live in the open air—in a tent—on a verandah. Few patients, however, have the luck to escape drugging, especially with creasote *ad hoc* *gravis* *osore*, and many of them afterwards suffer from indigestion and a ruined digestive apparatus. "Stick to creasote" is the reiterated advice of the physician who has not tried the effect of the drug upon himself. "Creasote" drugging was no part of Brehmer's system, and it is hard to understand on what evidence creasote has become so popular. Creasote in sufficient doses may be a germicide, but on no mathematical

calculation can it be shown that creosote taken into the stomach in drop doses and absorbed can in any way injure a single tubercle bacillus. Yet those who do use it seriously imagine that in some extraordinary way these few drops distributed through the body in a state of enormous dilution may lessen the activity and toxicity of millions of tubercle bacilli often lodged in tissues and passages far removed from the remote chance of even momentary contact with the drug. As an antiseptic or germicide, creosote given by the stomach must be inert when it reaches the lungs. If thus diluted it can affect a tubercle bacillus, I forbear to think how, in a pure state, it may affect the mucous membrane of the stomach. I have records showing that the use of creosote has been disastrous; yet some physicians have no scruples in applying this strong caustic directly to the delicate structures of the stomach wall for months and years. Thus Brehmer's system means something more than open-air treatment, and certainly stops short of creosote treatment. What a shocking burlesque of Brehmer's system our local physicians practise! They hustle off their patients into the country, so that they may escape the pernicious influence of the health-giving climate of the coast, and enjoin them to suffer the heat and dust and discomfort of the Australian bush, as well as the unsuitable food and bad cooking inseparable from it—provided only they take enough creosote to ruin their digestion for all time. A few may survive the horrors of such exile—many revolt—and all would do far better in a decent climate, with decent food and cooking, and in decent society. No physician practising these antiquated methods has dared to publish the results of his experiences, and yet many have dared to criticise my methods and my belief in the suitability of the sunny climate of the coast of New South Wales for the treatment of pulmonary tuberculosis. There has been much glib talk about sanatorium methods, as though sanatorium treatment had been demonstrated to be the only and the best treatment. In fact, the sanatorium for the poor at Westworth Falls is the outcome of this glib talk. It appears to me to be somewhat of a paradox that this system, which is claimed to be indispensable for the poor who suffer

from consumption, is not equally necessary for the well-to-do. Our self-appointed authorities in Sydney have exploited the charitable instincts of the wealthy and well-to-do, in order to provide sanatorium treatment for the poor, but do not consider it worth while to provide their well-to-do patients with those very means of treatment which they consider indispensable for the poor. If I had unbounded faith in the power of sanatoria to cure consumption, I should have urged long ago the establishment of sanatoria for the well-to-do. With our physicians in Sydney, a trip to the country is the panacea for their well-to-do patients—for the poor there must be sanatoria. It is quite clear to me that, as our physicians do not advocate sanatorium treatment for their rich patients, they should find it hard to convince their rich friends that sanatoria play a very important rôle in the treatment of consumption.

The treatment of pulmonary tuberculosis by climate has had a long trial. Hippocrates believed in change of air; Celsus recommended climate, sea voyages, and the inland climate in summer; Galen sent his patients to the mountains and recommended the milk cure. Many of our physicians mark time to the ideas of the ancients, and the value of these haphazard methods is written large in the black letters of the statistics of every country. Climate *per se* as an important factor in the treatment of pulmonary tuberculosis is now being discredited and discarded in the countries of the Old World. Else, how does it happen that sanatoria are being built in every sort of climate in England, Scotland, America, and Germany; while France, of all European countries, stands almost alone, for she has hardly deigned to consider the wisdom of spending large sums of money upon sanatoria for the poor. One of our authorities in Sydney boasts of the value of mountain air with one breath, and in another breath praises the results of sanatorium treatment in a hospital near London, where there are certainly no mountains. He even stated at a public meeting, at which I was present, that "75 per cent. of the cases at this sanatorium had been cured." I at once retorted that, if this were true, we should seriously think of sending our patients to this sanatorium, for we could not hope to obtain such results. Such statements are not

only grotesque, but cruel and dangerous to the cause he advocates. High authorities even contend that sanatorium treatment is independent of any climate, and should be carried out as far as possible under those very conditions of climate under which the patient has to live his life and earn his livelihood. This manifestly applies to the poor artisan who can barely afford to live in his own home. For such "hewers of wood and drawers of water," who form the largest section of society, sea voyages or constant changes to the country or mountains are mere dreams that can never be realised, because they cost both time and money. Yet many a time a lodge doctor advises his lodge patient to take a sea voyage or spend a year or two in the country.

In discussing the value of sanatorium methods, I do not desire to estimate the effect upon those well-to-do people who can devote many months in every year, and even a lifetime, as well as a fortune, in an earnest search for health—too often a "Will-o'-the-Wisp"—by means of sanatoria. Those who have the time, the money, and the patience to give to this object deserve every encouragement, and may be successful in their quest, but there are hundreds and thousands who have been sorrowful witnesses of the elusiveness of such methods. For a year or more there is apparent success which deceives everyone, even the physician. A relapse occurs with a recovery not so complete, and thenceforward the progress of events is ever downwards, perhaps still with some short respite, until even the physician has to despair where a few short years before hope had reigned triumphant. There are tens of thousands of people every year who have been eye-witnesses of these sad family histories. There is some consolation in the thought that faith in sanatoria, and pertinacity in exploiting its methods, may bring a good reward to 30 per cent. or 40 per cent. of those who trust this remedy. But it is surely no trifling matter that failure is stamped in big letters upon 60 per cent. to 70 per cent. of the cases that trust to sanatorium methods. At all events the great majority of the sufferers have not the means to indulge in this costly method of treatment. *The virtue of this system is*

not great when it can be applied only to a very limited number and falls in 60 per cent. to 70 per cent. of the cases. It is no exaggeration to say that in England there are at least 150,000 persons who need succour. If their only hope of salvation lies in the exploitation of sanatorium methods, their doom is sealed. No scheme for the exploitation of sanatorium measures can cope with this colossal task. If anyone replies that they cope with it in Germany in this way, I can only request those who hold such a view to study carefully and in detail the colossal work of the national insurance societies of Germany—their concrete objects and their results as published in the analyses of Engelmann and Hamel and Weicker. They will rise from the study of these great works both wiser and sadder men.

All must admit that it is very difficult to consider dispassionately and estimate accurately the value of any system of treatment in a disease so variable in its nature and course as pulmonary tuberculosis. The erroneous idea of olden times that consumption is the pitiless effect of some constitutional taint tended to encourage a fatalism—a very lethargy of ignorance and indifference—that has paralysed all rational effort for generations. Even now the medical profession seems but half awake, and medical men, who dogmatise upon the great benefits to be derived from sanatorium methods without a clear grasp of what such methods can do and cannot do, are but blind men leading the blind. A peculiar mental complacency and mental inertia may even afflict medical men. Would that one could shake authorities out of this deadly complacency so that they might at least carefully study the meaning and results of the economic and rational methods of the sanatorium, as applied to the poor, before they applaud and vaunt their efficacy. I should be the last to disparage or condemn the methods of the sanatorium merely because they were not perfect, but it is high time that, in the interests of truth and humanity, its shortcomings and failures should be as clearly recognised as its advantages and its successes are freely advertised.

Sanatorium methods are faulty and uncertain, and therefore it is our duty to seek and find a way to better results. Even

the laity have come to trust in the efficacy of sanatoria upon the somewhat glib assertions of medical men, and it is neither politic nor fair to mislead the laity upon such a vital national question. Hundreds and thousands of witnesses in England alone can testify that the sanatorium system, when put to the test on the confident advice of medical men, has signally failed.

Temporary results are common enough, but such results often follow no treatment at all, but permanent results are not by any means easy to secure. It is to me an awful mystery how any physician of wide experience in this disease can venture on the statement that the disease is curable; indeed, some physicians would almost persuade us that it is easily cured. It certainly would be easily cured if residence in a sanatorium even for six months would cure it. Many a patient is told that if he lives in the country or lives in the open air all will be well with him. Those who talk thus must rue it some day, for the germ of tuberculosis is particularly hardy and resistant and not so easy to kill, though it may remain dormant (quiescent, latent) for many years. The activity of the germ in tissue is such a variable quantity that no one can speak with any certainty of "cure" unless it has been shown by means of tuberculin that there is no evidence of such a dormant or latent tuberculous process. If, therefore, tuberculin is not used, we must remain in the dark as to the true state of the patient. *Further, these temporary improvements are so extremely common that we must discard altogether the evidence of results that have not been subjected to the test of time.*

The sequel to these temporary improvements—temporary arrest of the disease, etc.—is but a long, wearisome, disappointing chapter of relapses, terminating sooner or later in death—certainly in the majority of cases. We have a sanatorium in New South Wales supported by voluntary subscriptions, and at the end of each year the results of treatment are tabulated. Such results, neither controlled by test doses of tuberculin some months after, nor subjected to after-examinations, are absolutely worthless as evidence of the value of sanatorium methods in curing the disease. Yet

these are the records which the medical profession allow to mislead people. When I proposed that each year the cases treated in this institution should be examined in order to see whether the improvement was maintained for one year, two years, three years, etc., the authorities would not hear of it. Further, it is a rule of the institution that no patient can have more than one course of treatment. This also militates against any accurate valuation of the methods of the sanatorium. *Without such after-examinations, it is impossible to give a genuine value to sanatorium methods.* Many other diseases run a latent course, notably hydatid disease, even appendicitis. Because hydatid disease exists but progresses so slowly that it causes no symptoms, because appendicitis does not immediately cause severe symptoms, and runs a latent course, does the surgeon consider the disease to be arrested or even cured? Yet such a term is verily applied to so-called quiescent forms of tuberculosis, even when a test dose of tuberculin would reveal anything but latency. Accordingly, though the disease may be described by our sanatorium authorities as "arrested," in another few months, or later still, the disease may be rampant.

Often, indeed, all the improvement obtained by the sanatorium methods has been the abatement of the mixed infection, the specific agent of tuberculosis still lying in the tissues, little affected by the agencies of the sanatorium system. It is this abatement of a mixed infection that deceives the physician and tempts him to dream that the tuberculous process has abated. Again and again this temporary improvement is but a prelude to very bitter disappointment. *In the sanatoria for the poor there are very obvious reasons, apart from the mixed infections, for these temporary improvements.* Those who enter the sanatoria have been manfully maintaining a severe struggle for mere existence, working eight, ten, or more hours a day, ill-fed, ill-clothed, ill-housed, ill-attended. Is it a great miracle that a man who has been working from morning to night, living on bread and dripping, and breathing the air of a small, badly-ventilated room, should improve as soon as he rests

all day on a lounge, is fed every day on the best of foods, and breathes the beautiful air of the mountains or plains? Of course, he gains weight, and strength, and energy. But he may do all this and yet the disease itself persists—ready at any time to renew its attack, and slowly emaciate and exhaust the victim. Such temporary results are far from satisfactory, and, the world over, men have allowed themselves to be deceived by these transient successes until the inquiring mind of Germany—especially through the Imperial Board of Health (Engelmann and Hamel) and Weicker (the pioneer of a scheme for dealing with the disease among the poor)—set to work to inquire into the after-history of cases that had been treated in sanatoria. These after-examinations, made at intervals of one and more years after the patients have left the sanatorium, have shown that year by year the improvement vanishes, and at the end of four or five years after sanatorium treatment 30 per cent. to 60 per cent. of the persons so treated are dead of pulmonary tuberculosis. In Germany such after-examinations are the easier because the enforced system of registration of domicile helps one to track out the cases. In England and elsewhere the investigations would not be so simple, but until such investigations be carried out systematically we can but acquiesce in the conclusions based upon such investigations in Germany.

In 1903 I pointed out that English experience could not help us in determining the value of sanatorium treatment, because the sanatoria for the people had not existed long enough to supply the facts, and there had been no attempt at after-examinations. My assertion was scouted in 1903; in 1905 Dr. Ransome made the very same statement. Surely what was true in 1905 was doubly true in 1903. The whole gist of my argument is that we must have recourse to the experience and results of the People's Sanatoria in Germany if we wish to form a sound judgment upon the value of sanatorium methods; and even the experiences and results of these German sanatoria may lead us woefully astray unless these results have been checked and controlled by systematic after-examinations.

Let me repeat that I am discussing the value of sana-

torium treatment for the poor. For well-to-do people sanatoria do some good, sometimes much good, prolonging life rather than curing the disease. Such people can pay for the best advice, and it is their own fault if they do not purchase the best article. So long as fads, and fancies, and prejudices exist people may pay for them, and it is not possible to protect people from self-deception or imposition. This man abhors the idea of having a needle thrust under the skin; that man fears the injection may give him a disease; another hates new-fangled notions; the imagination is given free play, and unheard-of possibilities answer to its magic call. With the poor man there are no such difficulties. His faith is simpler and truer. As his health is the potent factor in maintaining the integrity of the family and home, he is ready to trust those who know far more than he does, and I have found that he is even ready to take risks. The conservative gentleman is fastidious, case-hardened in his prejudices, and "would rather bear those ills he has than fly to others that he knows not of." It is surprising, too, what an amount of idle and irresponsible chatter there may be among laymen upon the value of any system of treatment. It took me fully five years of laborious study and work to form an opinion on the very difficult question of the treatment of consumption. A layman is ready to come to a conclusion because he had heard that some friend of his had been cured by this method or that method. The poor man has a greater respect for constituted authority, and is less often the victim of humbugs than the intelligent and cultured gentleman. Of course, the humbug lays himself out for the wealthy, and ignores the poor for obvious reasons. I have abundant reason to be thankful to the poor, for they gave me my opportunity. Again and again the rich rejected my advice, and some have suffered. The poor took risks in the hope of better things, and my records embrace a very large number of individuals in the humblest stations in life. These records may be allowed to speak for themselves.

In Germany sanatoria for working men exist on an economic basis from the outset; not on a sentimental or humanitarian basis. By wise social legislation the artisan is

compelled to insure against sickness, and the great Imperial Insurance Societies of Germany have a direct interest in maintaining the industrial efficiency of the working unit and, perhaps, at the same time the fighting efficiency of the unit. By this system of State-enforced, not State-supported, insurance against accident and sickness, the individual workman is compelled to pay to the General Fund to which also the masters or employers contribute. Thus those who receive the benefits of these Insurance Societies—and sanatorium treatment is one of the benefits—have also to pay for it, at least in part. Every domestic servant must insure in the National Societies of Germany. These Societies, in dealing with pulmonary tuberculosis among wage-earners, devote much thought and time to the detection of the disease in its initial stage, because this is the most economical way of dealing with the disease. In about 65 percent of these cases, the disease is so early that tubercle bacilli are not to be found in the sputum, and in such cases the diagnosis is made certain by means of test doses of tuberculin. In N.S.W., hardly a case enters the sanatorium that does not show tubercle bacilli in the sputum, and, as I know to my chagrin, if a case does not show the presence of tubercle bacilli in the sputum, it is likely to be rejected. One such case of mine, in which I had already discovered tubercle bacilli in the sputum, was rejected after I had selected this very case for a clinical lecture on the early diagnosis of pulmonary tuberculosis. Accordingly our own sanatorium at Wentworth Falls is dealing with a less favourable class of cases. Indeed, under our present social system, the treatment of pulmonary tuberculosis *in an early stage* (according to German and not English or Australian ideas) is almost impracticable, because poor men, receiving daily wages, will not give up work when the disease is in such an early stage that it hardly affects their general health, and does not seriously impair their industrial efficiency. In Germany they manage these things better and compel the workman to seek treatment in the sanatorium as soon as the early signs of disease are detected, especially by means of tuberculin. One of the worst, the most dangerous and most treacherous features of tuberculous disease of the lungs, is

that often a man can work quite well, even when the disease has reached a relatively advanced stage; even when the disease is in the third stage the sufferer may be an efficient workman according to the German definition. It is a serious matter of national importance that there are no enactments on the statute-books of England or Australia that compel a workman to insure against the day of sickness. Friendly societies on a voluntary basis cannot pretend to deal with the treatment of consumption among the poor—at any rate by means of sanatoria. When the poor man ceases to work his income ceases and domestic misfortunes begin. Moreover, once pulmonary tuberculosis has reached the home of a poor man, this disease, like a vampire slowly spreading its wings, casts its blighting shadow in ever-widening circles. The father or the mother may be the first victim; and sooner or later other victims become enmeshed in the toils of this deadly family scourge. While the man, upon whom the family depends, can work, he will and must work, and in England and Australia more often than not the disease has passed beyond the hope of any remedy before he yields to the necessity of trying sanatorium or other treatment. So with us sanatorium treatment cannot be expected to yield results equal to those obtained in Germany, and even in Germany success is but partial. However, even if every man in the early stage of the disease were ready to take advantage of the sanatorium system, it would not be possible to provide enough accommodation for one-tenth part of the contingent afflicted with the disease in an early stage. Even in Germany, which provides for 30,000 cases every year at a cost of many millions of pounds upon building and close upon £1,000,000 a year for maintenance, there are nearly 200,000 cases in all stages which cannot have treatment, and the net result of treatment is that after a lapse of five years 60 per cent. of those already treated have succumbed to the disease. Hence it would seem to us that the sanatorium system, as a means of dealing with pulmonary tuberculosis among the poor, is at best a poor thing, often inapplicable, often inadequate and always expensive. With us the sanatorium system fails, (1) because men and women suffering from disease in the early stage are both

to seek refuge in the sanatorium, and there is no law to compel them; (2) because the results are not permanent, and (3) because it is far too costly to be of general application.

Let us now examine the evidence obtained from the experiences of German sanatoria for the poor, in order to see whether, after all, it is worth while in England to apply the same method *under less favourable conditions*. Even in Germany, where the system of sanatorium treatment has reached a climax, such as has not been approached in other countries, the net result has been that at enormous cost of money and labour, and by means of a colossal piece of State machinery, 30,000 to 50,000 out of an army of nearly a quarter of a million suffering from pulmonary tuberculosis are relieved from the stress of their disease for a limited number of years. Let us not misread and misunderstand the purpose of this treatment in public sanatoria. The system of sanatorium treatment for the artisan is based on economic principles, and the economy is manifest if the artisan, after treatment, maintains his industrial competency for a period of three years. For the insurance society and its economics it is enough if at the end of three or four years all those who have had sanatorium treatment are dead. Looking at the system from the points of view of the insurance society, the system is an economic success. But from the point of view of the *individual* artisan it may mean anything but success. In the majority of cases the individual dies of the disease in four or five years. Thus the net result of sanatorium treatment for the poor man is that in spite of his disease he is able to work (*arbeitsfähig*) for two or three years longer. The poor artisan is not cured of his disease, but is merely improved for a few years as a working unit. I refuse to believe that we have reached finality in securing no more than this economic success. He who is satisfied with these results of sanatorium treatment among the poor is lost. Nevertheless I would add that, although the sanatorium system hardly touches the fringe of this great problem, and is anything but an ideal system, it has some advantages, and may be almost indispensable in certain phases of pulmonary tuberculosis, especially when complications such as mixed infections, hæmorrhage,

and even pleurisy of an acute type have developed. It is not the value of sanatoria under these special circumstances that is open to criticism, but their great cost, especially when they are exploited in routine fashion for any and every case of pulmonary tuberculosis. I have proved that in the early stage the disease can be treated at home without even the sacrifice of the daily wage—with *tuberculin alone*—and the cost of treatment is within reach of the artisan, if special dispensaries are created for this specific object (see address to Sydney Municipal Council). In the later stage, when there is fever or great loss of vitality and tissue energy, sanatoria help greatly; but under these circumstances three or four weeks in a sanatorium is generally ample. If, therefore, my ideas were given practical form, it would mean that, *with a given sum of money*, one can do fifteen to twenty times as much good as the methods in vogue can do, and the good results would, beyond a doubt, last very much longer. My records demonstrate this fact beyond any question. Again, I say, if money is no object, people may enjoy sanatorium methods, for they certainly do good. But I have proved in very many cases that where sanatorium methods fail, tuberculin sometimes works wonders; and I am convinced that where sanatorium methods succeed, success by means of tuberculin is almost inevitable, and at a far higher level; because the successes with tuberculin have this great virtue—they last.

Before we consider the results obtained in the German sanatoria for the poor, it is necessary to criticise also the common notion that sanatoria *per se* play an important rôle in the prevention of consumption. With our present knowledge this notion should be buried in the scrap-heap. Sanatoria deal essentially with the disease in its non-infectious stage, so that the temporary segregation of early cases removes no dangerous source of infection. Again, the most infectious and most dangerous forms of the disease are rarely admitted into public sanatoria. It is therefore nonsense to suppose that institutions, whose avowed policy is to admit for a few months early cases, in which the risk of infection is minimal, and rigidly to reject the most infectious cases, can play a serious rôle in the prevention of tuberculosis. At any rate, I am

quite clear in my own mind that no government or municipal body, handling public funds, should devote any money to sanatoria until it has already made ample provision for the prevention of consumption in accordance with the orthodox principles of hygiene, and it is one of the essential principles of hygiene to attack the dangerous sources of infection—either by destroying them or by removing them out of range of danger. Sanatoria exist for the treatment of pulmonary tuberculosis, and it is not the function of governing bodies to undertake the treatment of disease except in so far as the treatment may be incidental to a policy of prevention. Thus in small-pox and plague, isolation of the sick is enforced, and therewith the government must also assume the responsibility of treating the disease till the period of infection is past. But no government enforces the isolation of those suffering from pulmonary tuberculosis, and therefore the government incurs no responsibility for treatment. If ample provision has been made by the central government for the prevention of consumption by the erection and maintenance of hospitals or refuges for the reception of the poor afflicted with the disease in a late stage, and municipalities provide measures for the notification of the disease with a view to the disinfection of premises known to be infected, then the treatment of the disease can be left to medical men either in the homes of the patients or in special wards in general hospitals or in sanatoria for the well-to-do; or in dispensaries for the poor. Thus the State government will have its hands full within its own clearly defined sphere of action, municipalities will have plenty to do within their own spheres, and there will be no interference with the medical profession; and there will be no overlapping of functions. It is not for State governments or municipalities to take in hand the costly business of providing and maintaining sanatoria for the treatment of this disease, though it may or may not be within the spirit of the law to support any and every system, especially the dispensary system, that aims at mitigating the intensity of the injury and suffering caused by this disease in every class of society. A last word upon sanatoria as a means towards prophylaxis. Much is often said of the educational value of sanatoria, and

I admit that the lessons of hygiene—of personal and domestic cleanliness—inculcated by the discipline of the sanatorium—especially with regard to the disposal of sputum and the proper way of coughing—are very valuable, but to my mind these lessons can be taught just as well by the properly trained district nurse in the homes of the poor. The proper precautions involve some irksome duties and, unless there is constant supervision and insistence, the victim of a long-lasting form of pulmonary tuberculosis soon lapses from grace. Even the pupils of the sanatorium, as I know full well, have often surprisingly short memories for inconvenient and irksome instructions, while the district nurse can daily urge the value and necessity of personal and domestic cleanliness and is bound to become in the future an indispensable handmaiden in the great work of preventing consumption in the homes of the poor. Again, the lessons at the sanatoria are very costly, while the lessons given by the district nurse are quite as effective and far cheaper. But from the point of view of prophylaxis tuberculin can claim advantage over every other system, for tuberculin properly used prevents the non-infectious form from becoming infectious and converts many infectious into non-infectious forms. All authorities agree that no method has such a marked effect in causing tubercle bacilli to disappear from the sputum as tuberculin. Hence tuberculin is also a valuable agent in the prevention of this disease.

At length we may turn to consider the value of sanatorium treatment for the poor as disclosed in the enormous labours of Weicker, Engelmann, and Hamel. Even in this work of the sanatoria tuberculin as a diagnostic agent is used to detect those early cases which are treated so successfully in the sanatorium. Without tuberculin mistakes would be inevitable. Thus by the use of tuberculin in diagnosis the value of the sanatorium is greatly enhanced.

Further, the sanatorium system starts with one immense advantage: it can pick and choose. In every sanatorium, in Australia as well as in Germany and England, the most favourable cases are selected for treatment, and chiefly by means of tuberculin. Thus by rejecting bad cases and

accepting many that are either not in an active condition or might get well without any treatment at all, the sanatorium has an enormous advantage over the poor practising physician who has to do his very best with all the cases, good and bad alike, which come for advice and treatment. As a practising physician in this plight I should hardly dare to make any comparison with sanatorium records, if it were not that I have been using a remedy which too many sanatoria have unwisely excluded. Sanatorium authorities admit that cases in the late second stage (II. III.) and in the third stage (III.) are not fit for sanatorium treatment. It is natural too that many of the failures of other medical men find their way to him who has long insisted that haphazard methods such as are generally practised in Sydney—not even sanatorium methods, but methods essentially based on the superstition that the climate of the mountains or of the plains of Australia has specific virtues denied to the climate of the sea-coast—are utterly untrustworthy. Scores of patients have proved the soundness of my contentions by the progress of the disease in their own bodies, when they have been hustled into the country and have come to me in the last throes of the disease. Naturally these cases belong to the statistical tables of the other fellow, though they may never appear there. I shall not damage my own results by inserting them. Accordingly I shall merely attempt to compare like with like, and shall place cases in the third stage of the disease in a class by themselves. Yet I must admit that my belief in tuberculin was profoundly strengthened by the extraordinary effects in a hopeless case in the third stage, which I treated about seventeen years ago, in 1891. A young man, *æt.* 26, came to Sydney and consulted a doctor who told him that there were cavities in both lungs, and he could not live three months. He came to me, and there was no doubt the first doctor was right. The patient was a walking skeleton. He had very abundant expectoration, amounting to many ounces of nummulated masses. I refused to treat him, fearing the catastrophe one read of in German and English journals in that year, but he was willing to take any risks. The discovery of the remedy had only been heralded

in the journals a few months before, and I had at once cabled for the tuberculin. I had already used it in half-a-dozen cases at Mr. Goodlet's Home, and had seen no catastrophes—indeed, nothing but good from it. In one case, the man had been under me at the Sydney hospital, and I had lectured upon him as an early case of pulmonary tuberculosis. He reminded me of this himself at Mr. Goodlet's Home. I gave him a test dose of '001 Old T. He reacted to 104. I treated him for some months. This man had been in the home for two years—in *statu quo*—and had never felt strong. Within five months of the first dose of tuberculin he went out a different man, having gained greatly in weight and recovered his energy, and eleven years after I heard of him as being absolutely well and at work. Accordingly, at the earnest solicitations of this hopeless case "from the country," I treated him, and he improved beyond my wildest expectations. Not only so: he returned to his work, and, as he was very poor, I taught him in 1891 to give himself the injections. I sent him injections for years, but I learnt too late that he knew more of the value of tuberculin at that time than I did. He assured me that *only the large doses did him any good*. Sometimes, to pacify him, I would send weak doses. In the face of European opinion I thought it risky for a young practitioner to yield to his piteous prayers for large doses. If I had given him tuberculin according to my later ideas, he would have lived longer. As it was he remained at his work for another six years—very much to my surprise. This history also shows that I am no new convert to the teachings of Professor Koch.

What lessons, then, may be learnt from German experience in sanatoria? No one in Germany can command greater respect upon the question of the value of sanatorium methods than Weicker, who has devoted his whole life to the study of measures for the relief of consumption among the poorer classes of society. Weicker began his noble work as an enthusiastic advocate of sanatorium methods *per se*, and, determined to arrive at the truth, he established and carried out a long and laborious series of after-examinations in the cases which he had already treated by sanatorium methods. These

after-examinations told the fatal tale. He discovered that of every 100 cases treated in sanatoria by rational methods alone, at the end of 1-1½ years 41, at the end of 2-2½ years 51, at the end of 3-3½ years 65, and at the end of 4 years 80 had lost their capacity for work or were dead. Such were the fatal figures revealed by his careful inquiries into the after-history of some *thousands* of cases that had received sanatorium treatment under his very eye. The death-roll was as follows:—

In 1-1½ years	45 %
In 2-2½ years	— 58·4 %
In 3-3½ years	65 %
In 4 years	80·7 %

Such figures may give a little satisfaction to those who were spared for a year or two the necessity of paying the tax for an employee's invalidism, but the victim has not much respite, if the result is merely a postponement of death for three or four years. Again, the aspect of sanatorium treatment is not encouraging when we read that of cases in Stage I.—

76 per cent. are unfit to work or are dead in 1-1½ years, 10 per cent. in 2-2½ years, 31·3 per cent. in 3-3½ years, and 55·6 per cent. in 4 years.

Such appalling records caused Weicker and others to pause and think. Weicker had carefully weighed sanatorium treatment in the scales and found it wanting, but, thank Heaven, Weicker has taken heart again in his noble effort to save the poor from the terrible results of this disease by becoming an earnest convert to tuberculin treatment. I regret, however, to find in his latest work (Wein, 1908) that he is still timid and fearful, for he approves and practises the use of very small doses. Perhaps my experiences may give him courage, and thenceforward he will obtain results that leave little to be desired. At any rate, both he and Moeller (formerly an utter sceptic, as I have reason to know, for I spent three days with him at Gorbardsdorf in 1899) are convinced converts, and among the most enthusiastic advocates of tuberculin treatment in Germany. If Weicker's evidence is not enough to gain a hearing for me, I would recommend any secker after

truth to read and inwardly digest (it will need a good digestion) the statistics of Engelmann and Hamel in their analyses of about 30,000 cases treated by sanatorium methods in various sanatoria in Germany. As soon as the searchlight of after-examinations is directed upon the temporary results recorded immediately after the course of treatment, the vaunted benefits of sanatorium methods gradually dwindle away, and the naked truth in all its sadness stands up in bold relief to tell us that the sequel to the long chapter of improvements is but a long, wearisome, and heart-breaking chapter of relapses, ending in death after a lapse of a few years in most cases. *Such a result, too, in cases that have been specially selected!* These publications are regular store-houses of information on this great question, and originally I intended to place many striking facts before my readers, but in substance these works merely show the same results as Weicker's investigations—demonstrating the conditions, limitations, and shortcomings of sanatorium methods.

Engelmann, as the officer of the Imperial Board of Health of Berlin, investigated the after-history of 20,878 cases that had been treated in sanatoria by the various Imperial Insurance Societies, and found as follows:—

In these 20,878 cases treated in

1897		1898		1899		1900
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and examined in 1901, the results lasted

3-4 years in 30%		2-3 years in 39%		1-2 years in 45%		1 year in 66%
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Weicker's inquiries in 2,469 cases show that in 35 per cent. of the cases the results lasted four years, in 41 per cent. three years, in 50.7 per cent. two years, and in 55 per cent. one year.

Certainly the records of the Hanseatic States and of Turban-Kumpf are better, but even Turban only claims permanent results in 38.6 per cent. after four years, and 38.8 per cent. of deaths. Turban, however, includes also results in the well-to-do patients, and the average duration of treatment was 222 days. The following table is also

instructive. The cases were treated in years 1896-1901, and examined in 1901.

Of cases treated in

1900,	52 %	were fit to work,	15 %	were dead		
1899,	48 %	"	"	26 %	"	"
1898,	56 %	"	"	41 %	"	"
1897,	51 %	"	"	48 %	"	"
1896,	45 %	"	"	62 %	"	"

Thus in two years 25 per cent. in four years 50 per cent., in six years 60 per cent. are dead, even though special care was exercised in selecting favourable cases for treatment.

If results approaching these had been my lot in practice, I should have been almost in despair. As it is, I turn with a sigh of relief and the buoyancy of hope to the other side of the page, where we shall consider this great national problem from the point of view of those who believe in the specific treatment of tuberculosis by means of tuberculin, either with or without the aid of sanatoria.

CHAPTER V

THE PROBLEM WITH TUBERCULIN AS A REMEDY

WE have already discussed the use and value of tuberculin in prognosis. Tuberculin in diagnosis is a very fascinating subject for investigation, and those of us—all too few—who have used tuberculin in diagnosis as a matter of routine and with no sparing hand can but marvel whence those fearful results had their being and origin, of which one heard so much from isolated individuals, whose names have since remained in oblivion. I knew of a man who had been treated with tuberculin by three doctors; one prepared the mysterious fluid, another man watched the proceeding, and the third inserted the needle. These doses were given every four hours. The patient had severe attacks of fever! and was alive thirteen years after to tell his story. Like other workers I had a very narrow escape more than once. On one occasion for some chance reason I postponed the dose for two days. The patient had some sudden accident in the abdominal cavity next day and died within twenty-four hours. If I had but given that dose what a fuss there would have been! Fortune favoured me that time. On two or three other occasions fever suddenly appeared when I had refrained from giving an injection. Of course, if men choose to generalise from an isolated incident they must err; but they must not expect others, who have more logical minds, to err also. After seventeen years of constant use I am able to say that I have not yet seen a single bad effect from one or several test doses of tuberculin. There may have been a severe reaction, but always a few days later the patient would volunteer the statement that he felt better than he had felt for some time, and almost invariably he would gain one or two pounds.

At first they generally lose a pound or two, but they soon regain this and an extra pound or two. One has not to wait ten days for this gain. It often occurs in half that time, which is further damaging evidence against the long duration of the negative phase as indicated by the opsonic index. I am sure that gain in weight under the same conditions is a far safer and infinitely easier index to the graduation of doses than Wright's opsonic index.

In all my work I have paid more attention to variations in weight than to any other single phenomenon, and I do not think it is safe to dispense with it. This gain of weight, too, may help to settle the diagnosis when the rise of temperature fails, or is of mild degree ($99\frac{1}{2}$ - $99\frac{3}{4}$). I consider a temperature of 100° to be conclusive, if accidental causes can be eliminated. If there is any doubt, it is best to repeat the dose, and then the most characteristic effect of tuberculin may be manifested—a still higher temperature, 101° or more, due to oversensitiveness to tuberculin (see nature of tuberculin reaction). In one interesting nervous case '002 Old T—the second dose—caused a temperature of $99\frac{3}{4}$. I declined to give a positive opinion, and the young patient refused to have another dose. He went to another doctor, and probably told him that I had said he had tuberculosis. I refused then as always to give a positive opinion on a rise of temperature under 100° F.

Tuberculin as a diagnostic agent gives no uncertain answer in two classes of cases. It detects tuberculous disease almost with the certainty of an electric current along a telephone wire, and excites a local response in the diseased area. On the other hand in many cases I have proved beyond all doubt that there was no pulmonary tuberculosis, when other doctors told the patients that there was disease and that they should go into the country for a year or two.

I saw one young woman who had spent a year and a half in a private sanatorium managed by a doctor in Melbourne. When she came to me I could find no evidence at all of pulmonary tuberculosis, but she had just the most typical physical signs of mitral stenosis one could possibly imagine. Her sister was a nurse, and I told this sister that her complaint

was heart disease and not lung disease. She was shocked to hear this, and determined to take her to another doctor, who absolutely agreed with my verdict. There was no escape from this conclusion, and yet this case had been treated for a year and a half for pulmonary tuberculosis because she had pulmonary hemorrhage, and I doubt not this case is recorded among the successful cases of this sanatorium. I also tested the two sons of a doctor in Queensland, who had already sent one of these boys to a sanatorium for a year without any benefit. When he brought the boys to me, he said they were just as bad as ever. I examined them, and found absolutely no evidence of disease. I also tested them up to 101 gm. Old T., and the result was negative. The father, a medical man, was staggered when I told him that neither of his sons had tuberculosis.

I also tested a woman who had been recommended by two or three doctors at the Prince Alfred Hospital for the Sanatorium for Consumptives at Thirlmere, where there were many advanced and infectious cases. Her husband had recently died of consumption. She gave a negative reaction to 101 cc. Old T., and was so pleased to hear the truth that she brought her daughter afterwards to be tested. Yet two doctors had no scruples in letting her go and live with advanced cases of the disease.

I have tested also several nurses who had been much with consumptive patients, and in every case but one removed a great load from their minds by proving that they were quite free from tuberculosis. I also tested a young woman who was on her way to the dry air for consumption. Her doctor said she had lung disease, and she took it for granted that she had the disease because she had lost weight. However, a friend of hers brought her to me, and I proved that there was no tuberculous disease of the lungs.

Another very peculiar case was sent to me by Dr. H. H. Examining the blood, I found well-marked lymphocytosis, and concluded that there might be either tuberculosis or syphilis. I excluded tuberculosis by means of tuberculin, and then sent back the case to Dr. H. H., as probably syphilitic. Antisyphilitic treatment had a rapid effect in dispelling

severe abdominal symptoms which had persisted for many months.

In another case there was a lesion on a knuckle of a girl, which looked to be tuberculous. I called in a specialist who agreed with me that it was either tuberculous or syphilitic. I excluded tuberculosis by means of tuberculin and refrained from adopting antisyphilitic medication till I had more grounds for the diagnosis. By simple treatment this chronic sore healed up and the girl has been well ever since. In two cases of bronchiectasis also I was able to exclude tuberculosis. In two cases of well-marked hæmorrhage from the lungs, I proved by means of tuberculin that there was no tuberculosis and the only other obvious diagnosis was hydatid disease. Two other cases came out to Sydney from England: one of them had been advised to give up a very good appointment and go to Australia, as his lungs were affected. He came to me within a day or two of his arrival, and finding no physical signs at all, I tested him and proved he had not tuberculosis. This case was a cruel illustration of a haphazard diagnosis. Surely before advising a poor man to give up a good position, *a few small doses of tuberculin might be given to save these unnecessary sacrifices.* Medical men may not seriously realise what such advice means. Would they be ready to sacrifice their own practice in obedience to such a random diagnosis? Another Englishman was sent out because he had developed a pneumothorax after a serious fall in the hunting-field, and Dr. K. F. was not sure whether there might not have been a localised tuberculous lesion. I proved by means of a test dose up to 101 grm. Old T. that there was no tuberculosis. In no less than sixty cases I proved that, although tuberculosis might have been suspected, it could be excluded with certainty by means of tuberculin. Many of these cases would have been sent up the country as cases of early tuberculosis and would be recorded as "cures." Unfortunately I have to be honest and exclude these cases from my list, although they are such easy cases to cure. But at least I have saved many from wandering about the country, as other doctors had advised, in search of a cure for a disease they had not got. On the other hand, needless to say, I have detected by means

of tuberculin many and many a case of tuberculosis which had not been recognised. This is of course a much more serious mistake, because if the disease is overlooked in the early stage, an irreparable injury may be done to the person who seeks your advice.

(A) *The Opsonic Index—its value in the Tuberculin Treatment of Pulmonary Tuberculosis.*

Perhaps nothing has helped so much to awaken fresh interest in tuberculin as Sir Almroth Wright's brilliant work upon opsonins and the opsonic index. The very name opsonin by its classical ancestry and its musical cadence has won a permanent place in the language of science, and whether we believe or disbelieve in the theory that called the term into existence, we have to thank the culture of its author for inventing such an extremely happy word. Though Denys and Leclerc first discovered the existence of the phenomenon, which this term explains, and Neufeld and Rimpau ventured upon the clumsy term, bacterio-tropic substance, Wright's new word "opsonin" enriches the language of science. Wright has done much more than make a new word. By his extraordinary skill in technique and his keen eye for the complex nature of biological processes he has cast a flood of light upon some of the most abstruse problems of biology. I am not in a position to express any opinion upon the practical value of his opsonic method in dealing with the common infections, in which staphylococci, streptococci and pneumococci play the chief rôle. I may, however, be allowed to say that his method—not new because Petruschky had already used it—is essentially specific, and, whatever may be the final judgment upon the value of the opsonic index in these and similar biological phenomena, it can hardly be questioned that Wright's method is a complete vindication of the method which Koch introduced for the treatment of tuberculosis. Indeed, since Wright has in very fact made use of tuberculin (T.R.) in order to apply his system to the treatment of tuberculosis, it goes without saying that Wright's method of treating tuberculosis is in principle Koch's method.

Accordingly whoever is convinced that Wright's method of treating tuberculosis is sound must at the same time confess that the real author of the principle is none other than Koch. Wright's method differs from Koch's method not in kind but in degree. Since, then, Wright's method is merely a modification of Koch's method, it remains for us to determine by experiment and experience whether Wright's modification is an improvement upon Koch's original method. The final test must be the test of experience, and the medical profession has abundant opportunities of testing the relative merits of these methods in the crucible of experience. Theories in themselves are at best but starting-points for further investigations. If after investigations the facts do not harmonise with our theories, it is idle talk to say "so much the worse for the facts"; our theories must be modified. The facts cannot change; our theories are changing every day. One has merely to read the investigations of Muir and Martin, Dean, Farrata, and especially the work of Hata in Wassermann's Laboratory, to learn that the so-called opsonin is not a simple body, but consists of two elements, which can be separated by the process of dialysis, resembling the ambocceptor, which is certainly specific, and a complement. As I have had the opportunity of working in Wassermann's Laboratory I know how thorough and trustworthy such results are likely to be. But I do not pretend to join in any discussion upon the nature of opsonin; I wish merely to give some evidence which has convinced me that however useful Sir Almroth Wright's work upon opsonins has been in stimulating research into many complex problems concerning the nature of immunity, Wright's method of treating pulmonary tuberculosis by his special system of exploiting the opsonic index as a guide in the graduation of doses of tuberculin is misleading, and can only lead to disappointing results. Again and again I have found that when none but small doses of tuberculin are given, there is improvement certainly, because in my humble judgment there is a rapid unpoisoning of the system, due as we suppose to the formation of antitoxin; but there have not been, and can hardly be, the more important changes—the formation of powerful

bacteriolysins, upon which the cure of the disease must ultimately depend. When we know how hardy and resistant tubercle bacilli are—surviving even for years in hard calcareous masses—it seems foolish to imagine that a few weak doses of tuberculin repeated at long intervals of time can produce any powerful bacteriolytic effect, even to destroying thousands and perhaps millions of tubercle bacilli such as occur in the second and later stage of pulmonary tuberculosis. *A priori* one would be disposed to discard Wright's theory, and any evidence favouring Wright's views would need to be very carefully scrutinised in a disease which in itself manifests so much caprice.

Now if the opsonic index means anything, any system which raises the opsonic index makes for immunity, and any system that lowers the opsonic index increases the disposition to the disease. Wright tells us that in localised tuberculosis small doses of tuberculin repeated every three or four days tend to lower the opsonic index and cause the "negative phase." Possibly that may be when small doses are given every few days. It is, however, quite certain that in all the cases in which I have had the opsonic index estimated, I have found that by gradually increasing the dose of tuberculin at intervals of three to seven days, varying with the degree of reaction, the opsonic index, far from being depressed, is raised far above the normal. In other words, by increasing the doses at short intervals up to very large doses *one produces an artificial immunity which is far in excess of the normal. Surely that is the very object of specific treatment by the process of active immunization.*

See Case 47, Stage II. of printed records. Treated in 1906. Opsonic index, 23.

He had had 3 cc. P.T.

And later 17 cc. T.R. = 7 mg. 3000 times as large as Wright's dose.

The effect of these large doses was to raise index to 23, and the immunity is so permanent that all hemorrhages have ceased (he had "terrible" hemorrhages), and in March, 1910, he is quite well.

In Case 30, Stage II. of printed records. Treated in 1906, the opsonic index was very low, .55, and yet immediately after the opsonic index was estimated, I gave this patient such rapidly increased doses of

T.R. that within five weeks a single dose of 2 cc. was given; and not once nor there any reaction. This man too has enjoyed good health ever since, and looks extremely well. Whatever can the opsonic index mean, when one can record such extremes! With this single exception I have found that after courses of treatment with tuberculin in large doses—say 2 cc. T.R., or 65 cc. to 75 cc. of P.T.—the opsonic indices have been high but variable—thus—

Case 45, Stage II. Treated in 1906. Of printed records at end

25.	V.	07.	5 cc. T.R. given.	
27.	V.	07.	5 cc. T.R. 1 mg.	
29.	V.	07.	Opsonic index, 1.4.	
21.	VI.	07.	12 cc. T.R.	
25.	VI.	07.	91 cc.	
29.	VI.	07.	Opsonic index, 1.	
30.	VII.	07.	Opsonic index, 1.5.	
34.	VII.	07.	17 cc. T.R.	
36.	VII.	07.	Opsonic index, 1.12.	
37.	VII.	07.	2 cc. T.R.	
21.	VIII.	07.	Opsonic index, 1.5.	
11.	IX.	"	"	1.16
9.	X.	"	"	1.78
17.	X.	"	"	.85
30.	X.	"	"	1.53
6.	XI.	"	"	1.34
14.	XI.	"	"	1.1

} Could not depress
index—steady and >
than normal.
Tested a year later.

Case tested in October, 1907—a year later—in reaction to .05 cc. Old T. March, 1909, absolutely well. Weight, 12 st. 7. Gained 27 lbs.

Case 72, Stage I. Treated in 1906.

T.R. up to 1.5 cc. (April 25th, 1907)

Opsonic index.

10.	VII.	07.	1.1
21.	VIII.	07.	2.34
17.	X.	07.	1.1
14.	XI.		.89

After treatment, weight increased to 9 st. 9—a gain of nearly 30 lbs. March, 1909. Tested: reacted—though no symptoms—had further treatment. Is to have further treatment.

Case 39, Stage II-III. Treated in January, 1907.

After treatment with P.T. up to 9 cc., gained 31 lbs. from start and afterwards.

Opsonic index, 1.25.

Tested a year after end of treatment.

No reaction to .05 cc. Old T.

Looks well, and has no symptoms. March, 1909. Still well.

Case 42, Stage II-III. Treated in April, 1907.

2.	X.	07.	5 cc. P.T. (Reaction, 92.6 deg.)
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Seven days later :

9. N. 07. *Opsonic index*, 1.6.
11. N. 07. 17 cc. P.T. 100 deg.

Tested 9 months later (July, 1908).

No reaction to 1045 cc. Old T.

March, 1909, very well. Weight 10 st. 12.

Gained 42 lbs. from start.

I had many other estimations of the opsonic indices made in patients in all stages of the disease, and even in late stages after tuberculin had been carefully given the indices were rarely under 1.

M. 07.

A hopeless case. Index, 1.44. In Stage III.

Mrs. W.—Also a case in Stage III, which responded well—though with severe reactions.

29. VI. 70. Index, 1.07.

March, 1909. Still alive, and coming to have further treatment.

Miss C. M. P. In Stage III.

27. VI. 07. 1025 cc. Old T. No reaction (in course of being tested).

23. VI. 07. *Opsonic index*, 1.21.

Miss D.—Enlarged glands relapsed after treatment with tuberculin.

11. IX. 07. *Opsonic index*, 1.07.

Miss McM.—A late case with mixed infection, improved by small doses of tuberculin; then mixed infection gained the day, and patient died early in 1909. I did not continue tuberculin.

10. IX. 07. *Opsonic index*, 1.19.

Miss C.—Also a case which progressed well under tuberculin until in 1908 a mixed infection supervened; she had an acute abscess of tooth, which was not successfully dealt with. This happened some months after I saw patient, and she died during my absence in June, 1908.

9. X. 07. *Opsonic index*, 1.7.

Miss L.—A hopeless case of generalised tuberculosis, involving lateral passages as well as lungs. Had a few doses of tuberculin.

7. X. 07. *Opsonic index*, 1.3.

She died soon after.

One can thus infer that doses of tuberculin repeated at intervals of three to seven days and increased with care tend to raise the opsonic index to and above the normal standard. If therefore the opsonic index means anything, it clearly proves that the system of dosage which I have used favours immunity; but the opsonic index itself is so liable to variations from trifling disturbances that it is not a trustworthy guide in the gradation of doses. By ignoring the so-called "negative phase," I believe that I have obtained results that I

could not have obtained by Wright's system of dosage. Not yet have I seen any records of successful results by Wright's methods in *advanced cases* of pulmonary tuberculosis. Even in the second stage of the disease, I have had at most four deaths out of seventy-four cases that have been treated since the year 1902—seven years ago. One of these cases relapsed after a period of six years, during which he had led an active, useful, and healthy life in the city of Sydney. He was so well that he foolishly disregarded my advice, and did not think it was necessary that I should test him.

In the second case the husband admits that I am not to blame. This patient also was free from all symptoms for five or six years, and unfortunately was *not tested* in the interval.

The third case died of a septic infection from an abscess in the mouth. Before this occurred she was doing well.

Unfortunately these three cases died during my absence from the Colony.

The fourth case died last week, and I am not yet sure he died from tuberculosis. I saw him last three months ago, when he seemed to be fairly well. What happened that he did not send for me recently I have no means of knowing.

Nevertheless four deaths in seven years out of seventy-four cases is a record of which I need not be ashamed.

Thus by Wright's own method it seems to be proved that large doses stimulate the mechanism of immunity and help to produce those bodies (antibodies), antitoxins, bacteriolysins, etc., by which the injurious effects of the tubercle bacillus are step by step reduced to a minimum; and finally even the bacillus may be wounded and killed in spite of its dense if not impenetrable armour.

As striking illustrations of final success in spite of large doses and even severe reactions, the following cases may be specially mentioned:—

Stage I.—Nos. 1, 3, 16, 20, 25, 31, 42, 50, 10, 67, 70, 71, 72, 73, 76, 77, 78, 84, 90, 99.

Stage II.—Nos. 5, 8, 11, 12, 13 (still well in 1909 in spite of very severe reactions), 37, 40, 42, 44, 45, 46, 47, 48, 57.

Stage III.—Nos. 15, 18, 29.

According to *newspapers* in printed records at the end of this work.

(B) Treatment by means of Sanatoria with Tuberculin as a remedy.

Infectious diseases of specific origin are the result of an affinity existing between the specific cause or its product or both and the tissue elements of the man or animal. The result of the disease depends on the action and reaction of these two essential factors. If the specific cause is very virulent or very abundant, it overpowers the vitality of the invaded tissues and destroys the organism. On the other hand, the resisting energy of the tissues may overpower the agents of disease and either destroy them or neutralise their dangerous products. Tubercle bacilli, the specific cause of all forms of tuberculosis, manifest a remarkable affinity for certain parts of the human body, notably the lungs, and the result of tuberculous disease of the lungs depends on the virulence and number of tubercle bacilli and the resisting energy of the invaded tissues.

In the treatment of such an infectious disease there are two rational methods—one directed against the specific cause and therefore called specific, the other aiming rather at strengthening the resisting energy of the invaded tissues.

These two distinct methods of specific and general treatment should be combined if we wish to get the best results.

The specific method of treating pulmonary tuberculosis consists essentially in the use of tuberculin in its various forms, which helps to destroy the bacillus and to neutralise its toxic effects. This method is in fact a process of active immunisation entirely different from the antitoxic treatment of diphtheria, in which the antitoxin is supplied ready made. In principle there is no difference between tuberculin treatment for the purpose of curing tuberculosis and the use of Haffkine's fluid or similar agents for the purpose of preventing for a short time such diseases as plague, cholera, or typhoid fever. If, then, the method is advocated in order to protect a healthy person against a disease, which in a mathematical sense he has a very small chance of contracting, it is not unreasonable to urge the use of the method if it offers even

a small chance of cure to one who has already fallen a victim to the disease. Those who have used and advocated the use of specific prophylactic fluids containing germ products for the prevention of plague cannot with any show of consistency oppose the use of tuberculin, which is, after all, merely a vegetable product of the living tubercle bacillus. Many are, in their ignorance, guilty of this strange inconsistency. On the other hand, I may disarm some opposition by saying at the outset that the *extremous* use of tuberculin is not the one only indispensable means of curing tuberculosis, even in an anatomical sense. We know of no disease in which death is inevitable. Always some individuals are ordained by nature or strong enough to survive an attack of the most virulent disease. Plague does not kill all its victims; nor yet tetanus or anthrax. This power to resist lies in the tissues. Let us go a little farther. This power to resist depends upon the ability of the tissues to produce a sufficiency of antibodies which either kill the specific agent or render its products harmless. It is too often forgotten that the very and only cause of the production of these antibodies in the tissues is the specific cause of the disease. *The disease works out its own cure by means of its own agent.* Not only the disease, but even the mechanism of immunity that cures it, are both the results of the living germ through its specific products. Except in this way no infectious disease cures itself or protects against itself. Even when we seek to strengthen the energy of tissue so that the disease may be resisted, we are blindly assisting nature to work a cure in nature's own way. In pulmonary tuberculosis sanatorium treatment, open-air treatment, the rest cure, and other methods act by stimulating the system to evolve the mechanism of immunity in response to the action of the products of the tubercle bacilli in the tissues, the most important of which is tuberculin. It may be a shock to some of the advocates of sanatorium treatment to hear that they themselves, without knowing it, have been innocently vaunting the efficacy of tuberculin in the treatment of tuberculosis. On the other hand, those who advocate the artificial supplementing of nature's own direct weapon of defence are quite consistent in

upholding the value of other methods, especially sanatorium treatment. Daily experience tells us that pulmonary tuberculosis is a disease that too often fails to work out its own cure. Some fault in the mechanism of immunity allows the germ to continue its work of destruction or, maybe, to lie in ambush for months and years. The whole purpose of treatment is to stimulate the tissues to build up and finish the mechanism of immunity so that the tissues shall no longer be at the mercy of the tubercle bacillus. *A priori* it seems logical to stimulate the mechanism by means of the specific products called tuberculin. *A posteriori* other methods evolved through empiricism are vaunted as the *ut plus ultra* and specific methods are held to be superfluous.

One sees cases of pulmonary tuberculosis, even complicated with tuberculous ulceration of the larynx or epiglottis, get well of themselves without any special treatment, without sanatorium treatment, without injections of any sort, without local applications.

In the first place, early diagnosis is the key to successful treatment. In the early stages the lesion of active or latent tuberculosis may produce physical signs like those of obsolete foci, and tubercle bacilli may not yet have escaped from the tissues. Obsolete lesions are not tuberculous, but post-tuberculous. How can the physician attempt to unravel the mystery of these various lesions? If the tubercle bacilli have disappeared there can be no tuberculosis, and if there is no active tuberculosis there is no reaction to tuberculin. This is a vital point. Tuberculin alone gives the clue whether the lesion is active or inert. If the focus is inert it may harbour tubercle bacilli that will become active at some future time. Yet, if the tuberculin reaction fails, it is likely that the tubercle bacilli are so surrounded that the tissues can themselves destroy the bacilli. On the other hand, a reaction to tuberculin proves that the lesion is not inert. This reaction is a danger signal of the first importance. It warns the physician of a danger hidden from sight, just as the fog signal warns the sailor of a danger in a mist which the eye cannot penetrate. By the systematic use of tuberculin we may learn the true nature of many of those lesions which Naegeli too readily

assumed to be tuberculous. Many of these lesions may be merely passive—quiescent, but still ready to be kindled into activity by inflammations or other infections. Sad to say, these closed lesions thus neglected may become the source of the acute and rapidly-progressing forms that develop in early adolescence. Now, if these terrible disasters may happen, there has been no cure, even though the lesion is for a time closed and inert. By no other way than by the systematic use of tuberculin can we distinguish these latent cases from absolute cures; and if tuberculin is not used, no one has a right to use the term *cure*. If the tuberculin test is negative, and again after an interval of three months or six months again negative, there is some certainty that a cure has been accomplished. When the prejudice against tuberculin has died its natural and inevitable death, one of the most valuable uses of tuberculin will be found in determining the effect of treatment of any and every sort. Meanwhile, except by those who use the test, the term *cure* must be avoided.

Let us add that the Tuberculosis Congress in London, 1901, deliberately adopted the opinion that tuberculin was an invaluable agent in diagnosis. The education of English opinion needed ten years before the truth of Koch's original statement was recognised.

Sanatoria cannot dispense with tuberculin as a diagnostic agent, and gradually tuberculin is taking its right place in all the sanatoria of Germany. If tuberculin is not used, mistakes must occur. True cases of tuberculosis are overlooked, and, on the other hand, there is the risk of condemning a man to an expensive and irksome course of treatment for a disease which he has not got. In one year Weicker rejected forty-eight spurious cases already diagnosed by good examiners as early cases. In the eager chase after early cases, otherwise trustworthy examiners may condemn to sanatorium treatment many cases in which the diagnosis rests not upon trustworthy evidence, such as tubercle bacilli in the sputum or a definite reaction to tuberculin, but rather upon the subjective impressions of the observer as to the character of respiratory movement or auscultatory signs. The tuberculin test alone can save the profession from the

Yes-No diagnosis. But for the tuberculin test Weicker might have greatly improved his statistics by including the rejected cases, and ultimately recorded them as *cases of cured tuberculosis!* Christian Science can do as much for these cases as the best sanatoria. Let us take care we give no such chances to the Christian Scientists to show their skill. It is an easy off-hand criticism to offer that tuberculin in diagnosis may lead to the inclusion of cases of latent tuberculosis that may never further develop. The same may be said of many of the cases upon which the statistics of sanatoria are based. Who will dare to say that it will not develop if a reaction to tuberculin occurs? On the other hand, by the use of tuberculin I have saved many persons the expense and inconvenience of exile to the country, to which they had been sentenced by various medical men. Further, when one uses tuberculin for treatment also, if perchance tuberculin has given a doubtful or false indication, the mistake is soon remedied. Thus if the reaction be no greater than 99.5° to 100° F., and tuberculin is given for treatment, a constant absence of reactions with larger doses rapidly increased arouses suspicion. In my experience, with proper doses a temperature of 100° is positive. A rise of temperature to 99.5° to 100° is doubtful. *Tuberculin is quite harmless, if there is no tuberculosis.* Why, then, should we fear to use it in the cause of sanatorium or other methods of treatment, when it surely reveals the presence of tuberculous lesions? I have no hesitation in saying that tuberculin is an invaluable agent in the selection of suitable cases for sanatoria, and also a ready means of gauging the success or failure of treatment.

A more open question is the value of tuberculin as a curative agent. It is extremely difficult to measure the value of different methods of treatment in a disease that runs such a variable course. For any and every method of treatment successes will be claimed. The only test is the *relative of successes to failures*. If we investigate the statistics of sanatoria, we find that the successes to-day are the failures of to-morrow. Every medical man has had successes, but far more failures. Moreover, records are practically worthless, *unless they show that after an interval of time, say*

two or three years, the patient is the better for treatment. It is the tradition of sanatoria to publish results as soon as patients leave the institution. This pernicious practice must give rise to misconceptions and disappointments. Many of the apparent successes of to-day prove the certain failures of to-morrow. Still the after-history of these recorded cases year by year will show the truth about the results of sanatorium treatment.

Three months' residence in a sanatorium may be well enough from an insurance point of view, since it may restore the industrial competency of a workman for one or more years, but in three months one cannot expect to arrest the disease, and it is arrest of the disease which the workman is led to expect and desires. The workman wishes not only to be able to work, but to recover his health. In most cases the work of sanatoria is patchy and unfinished. Patients are discharged—improved no doubt—but with the disease still potential, if not active, and merely awaiting a favourable opportunity to reassert itself. My purpose is to bring forward reasons for advocating the use of tuberculin as a remedy so as to bring the work of the sanatorium to a higher level of efficiency and completeness, for the benefit of the unfortunate victim of pulmonary tuberculosis, and even of the whole community. The better the work of the sanatorium the better for the community, and at the present time the inmates of our sanatoria are denied the advantages of tuberculin treatment that are enjoyed by patients in some of the best German sanatoria. The ideal treatment is specific treatment combined with those general methods adopted in the sanatorium that are of undoubted value in increasing that tissue energy upon which the success of tuberculin treatment depends. Nevertheless, in very many cases, although treatment in a sanatorium may be useful, it is by no means indispensable. In the next chapter I shall proceed to show that, without any sanatorium treatment, tuberculin can achieve all the success one would desire in a large proportion of cases of pulmonary tuberculosis.

Years ago when I was making my early observations with tuberculin, I studiously avoided the introduction of factors

that might vitiate my conclusions. Accordingly I instructed patients to live under the conditions to which they had been accustomed. Having thus proved that the uniform improvement could be only due to the new factor—the tuberculin—I lost no time in adopting all measures that might reasonably be supposed to help in the arrest of the disease and the cure of the patient.

Strange as it may seem, I am the only specialist in diseases of the lungs in Sydney who has gone to the trouble of providing sanatorium treatment for private patients, even though I consider the sanatorium methods of subordinate importance. Other medical men who openly proclaim that sanatorium treatment is of primary importance have taken no steps to secure for their private patients that method of treatment which they tell the world is the best and indeed the only treatment. It is idle to pretend that the haphazard exile of patients to the country constitutes sanatorium treatment. I leave it to the over-zealous advocates of sanatorium treatment for the poor to explain why it should not be equally successful in their well-to-do and rich patients. Let those who wish to be enlightened upon the great virtues of sanatoria methods read Dr. Ransome's article in the *British Medical Journal*. He quotes Walther of Noerdach to this effect, that, if by cure we mean all subjective and objective signs of disease have disappeared, no more than 11·20 per cent. can be so classed, although improvement occurs in 70·80 per cent. of the cases. Ransome writes himself, "I think it may fairly be said, as the result of a large mass of Continental statistics, that only about 30 per cent. of patients discharged from sanatoriums for the poor maintain the capacity for work more than four years." He also writes, "In England sanatoria for the poor have existed too short a time [writing himself in 1905] to allow of statistics of much value being collected from them." I said the very same thing quite two years before—in 1903. Later Ransome says, "Exactly half the patients discharged have maintained the improvement gained for over a year, while half have got worse or are dead."

Professor Moeller, of the Belzig Sanatorium near Berlin, and a recognised authority on tuberculosis, eight years ago ad-

utter sceptic upon tuberculin treatment, is now one of its most earnest advocates. He tells us that in cases in which sanatorium methods have entirely failed, tuberculin has been proved to possess undoubted curative properties, and that while sanatorium treatment never cures in three months, permanent cures are often obtained with tuberculin. Strange to say, Dr. Moeller uses tuberculin mainly in cases in which sanatorium methods have failed. My criticism of this half-hearted policy is simply this: If tuberculin succeeds where sanatorium methods fail, why ever should tuberculin fail when sanatorium methods succeed? Experience proves to me that tuberculin never fails when sanatorium methods succeed.

In his first record Moeller showed that—

Sanatorium methods alone cured	109 %
Sanatorium methods and tuberculin cured	83 %

Arranged according to stages the results were—

Stage I.—Sanatorium methods alone cured	51 %
Sanatorium methods and tuberculin cured	75 %
Stage II.—Sanatorium methods alone cured	19 %
Sanatorium methods and tuberculin cured	30 %
Stage III.—No cases possible except as cures.	

According to Moeller's latest experiences, *sanatorium methods alone* may cure 25.30 per cent. of the cases of pulmonary tuberculosis in the first stage (German Board of Health classification), while *tuberculin* in his hands has secured the same result in 84.6 per cent. of the cases. I venture to say that when Professor Moeller uses larger doses or repeats the course of treatment, he will find that tuberculin will cure 90 to 100 per cent. of these early cases. In fact, Professor Moeller will agree, as I do, with our great teacher, Professor Koch, that "*tuberculosis of the lungs in the first stage can be cured with certainty by tuberculin*." In the second stage, which Moeller maintains is never cured by sanatorium treatment alone, tuberculin gives permanent results in 40 per cent. to 60 per cent. of the cases. Even in the third stage tuberculin sometimes yields remarkable results that cannot be approached by sanatorium methods. When these splendid

results are claimed for tuberculin by those who have had an extensive and prolonged experience with it, are we going to be so foolish as to pay any heed to the carping, surreptitious criticism of those sterile authorities who have the hardihood to express adverse opinions on a matter concerning which they have no personal knowledge?

In medicine, as in surgery, pioneers have often had to bear much unfair criticism. Experience is our great teacher. I do not know any great authority who has used tuberculin with the conditions and limitations laid down by Koch and found tuberculin wanting. On the other hand, I have seen no carefully compiled records that prove tuberculin to be either harmful or useless. We do not condemn operations because novices and those who have not learned their art make mistakes and fail. There have been terrible tragedies arising from various operations in their early history, but the operations have survived. The failures and tragedies associated with tuberculin are not the fault of tuberculin, but the fault of those "fools who rush in where angels fear to tread."

Failures may be the stepping-stones to high success; and the failures of to-day are converted by increasing knowledge and experience into the successes of to-morrow.

(C) *Treatment with Tuberculin alone as the Specific Curative Agent.*

Thus far we have considered the value of the sanatorium treatment of pulmonary tuberculosis in its various stages *without and with tuberculin*. Weicker, Moeller, and others who closely watched the after-histories of patients treated by sanatorium methods *alone*, came to the conclusion that these methods failed except in a minority of cases. But these authors have proved that by the use of tuberculin the value of sanatorium methods is enormously enhanced, because tuberculin has the great virtue of converting temporary into permanent successes. From a logical standpoint, we have still to consider what success can be achieved when tuberculin alone is used. This was the problem which I approached at the outset, because, seeing that the sanatorium is quite beyond the

reach of the vast majority of sufferers, I wished to discover whether there was any means of helping the poor, who can neither help themselves nor obtain relief at the sanatorium. I have come to the conclusion that in at least 80 per cent. of the cases in the first stage one can dispense altogether with sanatoria, that at least in 60 per cent. of the cases in the second stage sanatorium treatment is not indispensable, and that even in cases in which sanatoria utterly fail to do any permanent good, tuberculin can often bring the disease to a standstill, at any rate, for many years. These are the general conclusions at which I arrived at least eight years ago, and it was because I had the courage to express my opinions candidly in the public interests that I aroused the opposition of the whole of the profession in Australia. The profession was wedded to sanatoria, and in attacking sanatoria I was guilty of creating dissension in this family party. Then it was that, independent of Calmette and of Dr. R. W. Philip, of Edinburgh, I worked out the idea of establishing and maintaining a dispensary in the city of Sydney, at which early diagnosis and prompt treatment by means of tuberculin should offer to poor patients the relief which they could not possibly obtain at a sanatorium. By means of this dispensary system of dealing with pulmonary tuberculosis among the poor, I have every reason to hope that in the majority of cases treatment with tuberculin at the dispensary could be carried out without loss of employment, and, therefore, without the loss of the daily wage. This is a great advance upon any other system, if it can be applied with success.

In the results which I published in 1901 I had adopted tuberculin treatment pure and simple, because I did not wish to disturb the simplicity of the logical problem which I attempted to unravel—viz. whether tuberculin was a remedy for pulmonary tuberculosis. Every patient remained in or near Sydney, and lived his ordinary life. I had used Old T., T.R., and T.R. Emulsion (T.E.) in full doses; indeed, all the preparations which were magnanimously offered to a medical profession which, like the Pharisees of old, rejected the one means of salvation for the multitude of victims held tightly in the grip of pulmonary tuberculosis. In 1901 I reported twelve

cases of pulmonary tuberculosis in Stage I which had been treated by tuberculin. Out of these twelve cases I know nothing of two. Of the remaining ten, one had to go to Victoria before she had received full treatment. Under partial treatment she improved immensely, gaining 18 lbs. and feeling well and strong. I know no more. Another case, a young girl *æt.* 17, improved greatly; she lost her cough and phlegm, tubercle bacilli (formerly G. 89) disappeared from the sputum, but after several months' treatment she became very sensitive to tuberculin, and, in accordance with Petruschky's method, I advised a pause of six months. She went away to the country and felt so well, that, without consulting me, she took up nursing. Later, I learnt that she had been in effect prevented from having further tuberculin treatment by a medical uncle, who was an authority on lunacy, and had a strong prejudice against tuberculin treatment. As I foresaw, the disease relapsed, and in spite of the uncle's methods, the poor girl died about a year and a half after she left me. The father afterwards told me that he had regretted ever since that his daughter had been removed from my care and subsequently he sent his other daughter to me for treatment. In this latter case the result has been entirely satisfactory. I am therefore justified in scoring these cases out of my list, and may treat the second case as a "control" case. This case is the only case which refused my advice; and she is dead.¹

Case 1, treated in 1892, was quite well in 1903.

Case 2, treated in 1898, had a slight relapse, but in 1908 was well.

Case 3, treated in 1901, was well in 1907.

Case 4, treated in 1899, had a relapse in 1904-5, but after another course was quite well in 1907.

Case 5, treated in 1900, was well in 1907.

Case 6, treated in 1900, was well in 1908. This man had four "control" cases in his brothers. I abstract this account from my article in 1901:—"Mr. E., *æt.* 47, gave an ominous "family history. Four brothers, one after the other, had "succumbed to the disease. They had been under various "physicians for treatment. They had been sent to the

¹ 1908. Case 8 (fol.)

"country and treated in the usual way; but it was of no avail. The last survivor, having seen the failure of ordinary methods, came to me (from Melbourne) when the fatal disease showed itself in him, as in his brothers, by cough and expectoration, and general loss of health and weight. I examined him and found very evident signs of early disease. No tubercle bacilli found in sputum. I therefore gave a test dose, and 100 cc. Old T. raised temperature to 103° F. All doubt was at an end. He went under a course of T.R. up to 2 grms. The patient proved to be very sensitive, and the course occupied six months. During the course the patient had severe attacks of gastritis and enteritis, with diarrhoea. At one time the weight fell considerably, and I almost despaired; but ultimately the result was satisfactory. The cough hardly troubled him, the expectoration ceased, and he felt greatly improved. The improvement continued for a few months, and then the old symptoms began to reassert themselves. I gave him a second course. The second course he took very well; in two months one was able to give him the full dose (2 grms. T.R.). Again the improvement was very marked. Some months afterwards I saw him, and he seemed in perfect health. His weight increased from 11 st. 7 lbs. to 12 st. 5½ lbs., though during early treatment his weight was only about 10 st. 10 lbs. He told me that in the same time, from the beginning of their illness, his brothers had all succumbed to the disease. The treatment was carried out while the patient continued at his business, though he lived out of town. The second course hardly even upset him. Surely some great change had been worked in his system that he bore the second course so easily."

In 1908 this man enjoys perfect health.

Case 7 was treated in 1897 (Tb. G. 3), and was well late in 1907.

Case 8 was really in the second stage; severe mixed infection, sputum abundant, and tubercle bacilli in myriads (G. 9). He was treated in 1901, and remained well till 1907, when he had a relapse, with fever and hæmorrhage, and later laryngeal tuberculosis. This patient, in spite of my urgent entreaties, would not come to be tested. He had a very large

practice, and brought on his relapse by sheer overwork. He had to use his voice to straining point in his work, and to keep his worn-out body up to the mark he took far too much alcohol. It was inevitable that the body would break down, and in this case the breakdown occurred in the over-used organ—the larynx. He died in May, 1908, and I have little doubt that if he had been tested two or three years ago he would have reacted, and we could again have stepped in with tuberculin and prevented the catastrophe that occurred.

Thus, of eight cases treated from 1892 to 1901, seven cases are well, and one case died this year under conditions which show that tuberculin worked wonders for him. Of cases in the second stage, one case, complicated with laryngeal tuberculosis, survived nine years; two others, in the late second stage, survived respectively eight years and nine years; one died from a sudden hæmorrhage in five years; five others I have seen alive in 1907, treated respectively in 1899, 1900, 1901, 1899, and 1900. I know also that two cases in the third stage, treated in 1900 and 1901-2, were alive in December, 1907.

Such after-histories will compare favourably with any after-histories of cases treated by other methods. My own experience teaches me that the effect of tuberculin treatment, although always good—indeed far better than any other treatment—varies in degree and extent (of time). Beyond all doubt, so far from Koch recommending too large doses, as Wright, trusting to his opsonic method, maintains, the doses of tuberculin, recommended by Professor Koch, are hardly sufficient to do the work asked of the remedy. For many years since these early observations of mine I have been giving large doses of T.E., and more recently still of Old T. and P.T., with most encouraging results. I frequently give 4 grms. of T.E., and if this be not sufficient it is wise perhaps to try other forms, especially P.T. or T.A. My observations in 1901, in the light of later experience, show that T.R. is a very useful remedy in early cases, but may or may not be sufficient to prevent relapses. *There is no better way of testing the value of treatment than by its effect in preventing*

relapses. We cannot expect to know whether there are still living bacilli in the tissues, but we may be able to tell by means of tuberculin whether the bacilli are active. In cases therefore that have been treated by tuberculin, if the reaction to progressive doses—say to .005 grm., .015 grm., .03 grm., .045 or .05 grm. of Old T.—fails to develop in any one who a year before had had a full course of tuberculin treatment, it is fairly certain that either there are no germs living in the tissues, or their action has been entirely inhibited. At any rate I have proved in many cases that the reaction fails when all the clinical features and symptoms suggest that the disease has been arrested, while the reaction develops invariably when the symptoms and signs point to fresh activity of the disease. One knows that cases occur, in which, although tubercle bacilli are being expelled from the lungs, there are none of the usual symptoms of open tuberculosis—at most a slight cough and slight germ-laden expectoration. I have seen no such cases, but it is said that such a condition may continue for years and be compatible with apparent health. The lesson of such cases cannot help us until we know the effect of test doses of tuberculin upon them. Moreover, by means of tuberculin these open infectious forms can be converted into harmless, non-infectious forms—surely a "consummation devoutly to be wished."

Accordingly *I regard this system of gauging the effect of treatment by these test doses of tuberculin not less than nine months or a year after treatment as an invaluable adjunct in the treatment of tuberculosis by specific methods.* More than this, I hold it to be very sound practice to submit patients to such tests regularly every year. These doses cause no trouble, no pain, no disturbance of any sort, except in cases that have relapsed; and in such, this discovery of a positive kind is well worth paying for at the trifling cost of many purely negative results that are totally devoid of any risk. One knows how the surgeon argues who wishes to operate upon every case of simple catarrhal appendicitis. One may say that these cases left to themselves do not cause 10 per cent. of deaths. Accordingly the surgeon literally advises that he should be allowed to do ninety unnecessary operations with all

the attendant risks in order to reduce the number of deaths. The gain to the individual depends absolutely on the special skill that can reduce the risk of the operation to a minimum, but in any case ninety operations are performed which do no good. Am I, therefore, asking much in proposing that all patients who have been treated for tuberculosis by specific methods should be tested from time to time by injections that can do no possible harm in the hands of an experienced physician? In fact, until tuberculin is thus exploited in diagnosis, in treatment, and also in prognosis, we shall fail to make full use of this great boon to humanity. If I had had my way and tested the one case which after tuberculin treatment relapsed and died, I believe this patient would be alive to-day. But the patient had been well for so many years, and was so busy, that he did not think it necessary, and meanwhile tuberculosis laryngitis supervened. I am sure that *in the treatment of this disease, even with tuberculin, we dare not be over-confident.*

Thus I may claim in Stage I., after an interval of from seven to ten years and more, nearly 90 per cent. of successes. While I was using tuberculin, I scrupulously avoided sanatorium treatment. At most I told the patients to take good food and plenty of it. I did not send a single case into the country at any time, and ever since these patients have led the ordinary lives, not of invalids but of strenuous citizens. How many of the sanatorium successes are given this freedom? They are enjoined to be always in the country—to do this and not to do that—as though poor people had it in their power to choose the good only and reject the evil. I have therefore shown that in those very cases, which are supposed to be *par excellence* the cases for sanatorium treatment, much greater success can be achieved by means of tuberculin than by means of sanatoria, and at a very much smaller cost.

In short, while the rich and well-to-do may, if they choose, at the behest of their medical adviser spend money and time—even a lifetime—in seeking the will-o'-the-wisp health, now at this sanatorium, now at that, it is high time for people of ordinary means, especially the wage-earners of the community,

to know that even though they have fallen a prey to tuberculosis they may get the better of their disease and recover their health, more easily, more rapidly, more certainly, not to say at a far less cost of time and money and without loss of wages or work, by means of the remedy magnanimously thrust into our hands by the genius of Professor Koch,—a priceless boon to suffering humanity from the hand of the scientific spirit of Germany.

Beyond all doubt this remedy can be best brought within reach of the poor of cities by means of the dispensary system, which I fully discussed when I brought this subject before the Municipal Council of the city of Sydney as one of the aldermen. (See Address.)

Since 1901, when I first published my experiences with tuberculin, I have had no reason for excluding sanatorium methods, and I have adopted a mild adaptation of sanatorium treatment for all patients who could well afford these other methods. However, I do not consider sanatorium methods a *via* *quod* *non* to success. I have treated many scores of cases without any suggestion of sanatorium methods with complete success, and most of these patients belong to the class for which tuberculin treatment can be easily exploited at the dispensary and through the dispensary system. Among my patients were many servant girls (8), shop girls (5), other poor girls (8), nurses (3), many poor mothers, teachers in public schools (6), labourers (about 12), printers (2), butchers (2), actors (4), tram-guards (5), railway men (2), harmaids (3), hotel-keepers (3), clergymen (4), singers (3), clerks about (25), waiter (1), milkmen (2), fruiterers (2).

In all these cases they spent their time as they liked and went daily to their ordinary avocations.

It is therefore abundantly clear to me that the energy that is now waiting to be expended for the lasting benefit of the poor already in the thralldom of this prevalent disease should be directed along that channel which offers the best results at the least cost. Sanatoria are far too costly to bring much relief to the poor, and, besides being inadequate in so many cases, are decidedly inferior to systematic treatment by specific remedies. In my opinion the logical force of my argument is

absolutely unassailable, unless later workers can show that my results are untrustworthy.

I shall await with perfect confidence the verdict of any system designed to put my experience to the test of actual practice, but I fully recognise that it will not be possible to test the truth of my observations in a short space of time. It needs several years to prove the uncertainty of the inferior methods of the sanatorium. It must take a longer time to test the results of a method which is held to be superior. Although I know that public opinion is entirely against me, I still assert, on the strength of a long and laborious period of close and constant observation, that in a few years' time the specific treatment of pulmonary tuberculosis by means of tuberculin will be the recognised system of the scientific and conscientious physician.

This final chapter upon the results of tuberculin treatment must be read with the records of cases which have been treated by me since 1892. I admit that after the wave of condemnation that swept over us in 1893-1894, it was extremely difficult to find any who were ready to submit themselves to treatment. Such good things were promised by the advocates of sanatoria, and such bad things were said of tuberculin treatment, that the patients who came to us were chiefly the failures of other systems. We rarely had the chance of treating even a case in Stage II., and the methods of 1893-1897 were hardly suitable for Stage II.-III. and Stage III. In 1897 tuberculin T.R. (New Tuberculin) was given to us, and the early experiments proved it to be of value especially in simple, early, uncomplicated cases. However, I doubt if T.R. can often help us much in Stage II.-III. or Stage III. T.E. was then tried, and I continued to use T.E. for all cases that were at all advanced; sometimes, however, after some preliminary treatment with T.R. On the whole, however, I came to the conclusion that while these preparations were of great value in Stage I. and Stage II., it was questionable whether permanent good could be obtained in Stage II.-III. and Stage III. by either T.R. or T.E. Often it seemed to me that the Old T. was better than T.R. + T.E., especially when the disease was advanced. It was a great step forward when in 1904 and subsequently I began to

use P.T. As I have already pointed out, P.T. is useful in all stages, but especially in Stage II.-III. and Stage III.; and a previous course with the milder remedy P.T.O. is very useful, because this preparation is borne so well.

Method.—For very many years I have adopted the plan of injecting the doses into the forearm or outer part of the upper arm. The skin should be rubbed well with spirit, and strict asepsis must be practised. In two cases, in which I allowed a doctor and a nurse in the country to give the doses, abscesses appeared at the site of inoculation. Thereafter I taught many patients to give themselves the doses, which I used to send at certain intervals. In this way I was able to treat cases in New Zealand, Tasmania, Queensland, and in the inland parts of New South Wales.

As I found no serious trouble followed even well-marked reactions, provided they were not too severe nor too frequent, I did not use extreme care to avoid reactions. Indeed, I am convinced that an occasional reaction with fever often does great good, especially in the early stages of the disease, and in the earlier stages of treatment (see records). When no reaction occurs, I generally give another and larger dose in three or four days. If a reaction occurs, I increase the interval according to the degree of fever. Thus, if the temperature be 100° I give the next dose in five days, if 101° in six days, if 102° I wait a week or more, but I rarely wait more than seven or eight days, except in the case of the larger doses (2 cc., 3 cc., 4 cc. of T.E.). Moreover, if the temperature be above 100.5° I generally repeat the same dose. If the temperature be above 102° it may be wise to diminish the dose or to suspend the injections altogether for three or four weeks. After a fortnight's interval of time it is not wise to increase the dose, and it may be best to diminish it. After a month's interval the dose must be reduced to $\frac{1}{2}$ or $\frac{1}{3}$ of the previous dose. Then it is often an easy matter to proceed to larger doses rapidly—at intervals of three or four days. However, these details depend upon circumstances, and we should always bear in mind Koch's original warning that there must be no routine method, since each case has to be treated on its own merits. Again, it is not easy to say what should be the maximum dose in each

case. I think the body weight must be taken into account, and as a matter of principle I try and give as large doses as possible, provided the condition of the patient improves and he gains energy and weight. To a full-sized adult man I give as a maximum dose: 1 cc. P.T., 1 cc. Old T., 2 cc. T.R., 4 cc. T.E. I have used T.O., and T.O.A., and P.T.O. (T.O. for intravenous injection), but I am never satisfied with these alone. According to my experience these doses are not too large, if they can be reached without too severe reactions. It may take four months, six months, eight months, or longer to complete a course of treatment; but seeing that the treatment can be carried out without interfering seriously with a man's business or work, this question of time is of no great importance. If the course takes a long time, the intervals between the doses are also longer. (See special chapters.)

Besides the injections of tuberculin, I merely give rational instructions to the patient to maintain the tone and vitality of the tissues, and to avoid excitement, over-exertion, and excesses of all kinds. I am sure that alcohol very rarely does any good in pulmonary tuberculosis. Good health depends largely upon good food and good digestion, and good health must be maintained throughout the treatment. As for drugs, I have no belief in the drugs so much in fashion. I have not given a single dose of creasote, or substances of this class, and I have no intention of ever doing so. They may do great harm; I cannot imagine how they can do good.

PART V
RECORDS

PART V

RECORDS

INTRODUCTION TO RECORDS.

ONE cannot pretend to give a full account of several hundred cases, nor is it easy to condense into a few lines the observations of many months. In defining the stage, I have used mainly Turban's scheme of classification. By Stage I., however, I mean that the physical signs, if any, are limited to a small area, chiefly the apex of the lung, not below the level of the clavicle in front and the spine of the scapula at the back. By Stage I.-II. I mean that the physical signs extend rather lower, as far as the second rib. These stages are fairly definite and imply an early stage. If the disease has extended still further, say below the second rib, and involves scattered areas of small size in the upper lobe, it is Stage II. In this stage good results are still possible if the lesion is open, and mixed infection is not present. With mixed infection the expectoration increases in amount and the specific pyogenic organisms abound in the sputum. Then, also, softening is likely to occur, and if it be at all extensive, and especially if similar though less advanced changes are occurring in the other lung, I speak of Stage II.-III. The extent of the dullness and the degree of the softening determine this stage, and with these changes also the character of the adventitious sounds. The amount and character of the sputum help to define this stage. If the physical signs are observed over most of one lobe or over one half of two lobes and the sputum exceeds an ounce or two, Stage II.-III. is present. Any more advanced stage, in which the whole of the lobe is involved, or evidence of well-marked cavitation exists, is Stage III. It is relatively easy

to classify cases when the disease has not passed Stage I.-II.; but it is often very difficult to define the limits of Stage II., Stage II.-III., and Stage III. Cases of pleurisy, with or without effusion, I define as Stage I, unless there are other lesions elsewhere. The duration of the disease may help but may mislead. Stage I, or I.-II. may persist for a year or more, and on the other hand Stage III. may be reached in six months from the onset of symptoms.

Turban's classification of the stages of pulmonary tuberculosis is largely followed. He recognises three main stages (I., II., III.), with two intermediate stages (I.-II. and II.-III.). It will be convenient to include this classification as it appears in Weicker's "Beiträge zur Frage der Volksheilstätten."

TURBAN'S STAGES OF PULMONARY TUBERCULOSIS.

	Pathological.	Physical Signs		
		Percussion.	Auscultation. Breath sounds.	X-ray.
Stage I.	Bronchitis. Peri-bronchitis, scattered foci in one apex. No definite consolidation. (Often no tubercle bacilli in sputum.)	Normal or slight want of resonance over area of one lobe or one half of two lobes at most.	Wet, weak, sub-crepitation-like or broncho-vesicular over one or both apices.	Microscopically fine or medium over area of attack, none else.
Stage II.	Bronchitis. Peri-bronchitis, infiltration of slight degree over both supracardiac & subcardiac or marked infiltration of one lobe exclusively.	Diminished resonance over one lobe or some moderate loss of resonance over half of two lobes.	Wet to broncho-vesicular crackling over half chest.	Edges of medium quality.
Stage III.	All cases more advanced than Stage II.	Pronounced dulness may be symmetrical or not. (Checked post mortem.)	Bronchial Follicular Amplic.	Shaded even thick, etc.

Cases between I. and II. marked I.-II.
Cases between II. and III. marked II.-III.

EXPLANATION OF PLAN OF RECORDS.

In the subjoined tabulated form of records I have followed in the main Turban's scheme. It is, of course, impossible to do more than give a bird's-eye view. The plan is as follows:—

Name (A or B), age, weight (in ounces), family history.

1 — Stage of disease (I., I.-II., II., II.-III., III.).

2 ☐ if quiescent, —> if active and progressive.

3 Duration of illness.

4 State of nutrition: A=good, X=bad.

5 State of digestion: B=good, V=bad.

6 Pulse rate.

7 Temperature: N=normal, / 99°-100°/5°, F=(high) 101°-105°.

8 Tubercle bacilli absent or present according to Gaffky: M=mixed infection, S.P. I (streptococci, pneumococci, influenza). Amount of sputum.

9 Tuberculous complications: pleurisy, bronch.

10 Other complications.

11 Energy: capacity for work, 4, 3, or 2.

12 Treatment: previous, present.

Doses of tuberculin always expressed in cubic centimetres (cc.). 10 cc. or 100 minims $\frac{1}{4}$ cc.

STAGE I.

1902.

1	3
<p>sl. cr. 3½. gr. 10 lbs.</p> <p>No family history. Singer. Consumed for strained voice.</p> <p>1 R. I.-II.</p> <p>2 G.</p> <p>3 1 year.</p> <p>4 A.</p> <p>5 B.</p> <p>6 N.</p> <p>7 N.</p> <p>8 No Th. Resisted.</p> <p>9 Complete paralysis of right vocal cord; nerve involved in scar at apex of lung.</p> <p>10</p> <p>11</p> <p>12 T.R. up to 2 cc. (103°), then up to 1½ cc. with moderate reactions. Great improvement in general health. Cord always paralyzed. Well.</p>	<p>sl. cr.</p> <p>1 R. I.-II.</p> <p>2 —</p> <p>3 ?</p> <p>4 A.</p> <p>5 B.</p> <p>6 N.</p> <p>7 N.</p> <p>8 No Th. 1001 cc. Old T. 1001°.</p> <p>9</p> <p>10</p> <p>11</p> <p>12 Treated with T.E. to 25 cc.; then T.E. to 35 cc.; then Old T. to 100 cc.; then T.R. to 17 cc. (100°), wt. 9½ lbs.; then T.E. to 18 cc., wt. 9½ lbs.</p> <p>Some reactions:—</p> <p>1.v.01. 15 cc. T.E. 103°.</p> <p>1.v.02. 14 cc. T.E. 99°.</p> <p>2.v.03. 14 cc. T.E. 102°.</p> <p>1.v.05. 15 cc. T.E. 100¼°, and gained 1½ lb.</p> <p>Felt quite well.</p>
2	4
<p>sl. cr. 3½. 101. 10 lbs.</p> <p>Sister 8½ year ago.</p> <p>1 R. I.-II. &.</p> <p>2 —</p> <p>3 6 months.</p> <p>4 X.</p> <p>5 Y.</p> <p>6 N. 90.</p> <p>7 100°-101°.</p> <p>8 No Th. Reacted 1001 cc. Old T. 1001°.</p> <p>9 Pulse in left wrist, and in ankles.</p> <p>10 Later wt. 101½ lbs. and 101.</p> <p>11</p> <p>12 T.R. up to 15 cc., 1001°, many reactions, but quite well for 5 years.</p> <p>1107. Tested. Reacted. Second course P.T.O. to 9 cc., wt. 101½ lbs.; then P.T. up to 5 cc.</p> <p>3.vi.07. 35 cc. P.T. 101°. No doubt injected into veins. Shivering ½ hour after done; no swelling in arm; well in evening.</p> <p>28.vi.07. 5 cc. P.T. 98¼°.</p> <p>Well.</p>	<p>sl. cr. 101. 10 lbs.</p> <p>1 R. I.-II.</p> <p>2</p> <p>3</p> <p>4</p> <p>5</p> <p>6</p> <p>7</p> <p>8 No Th. 1001 cc. Old T. marked local reaction. 1001°.</p> <p>9 1004, 100¼°, and great local reaction.</p> <p>10</p> <p>11</p> <p>12 1000 T.E. 99½°.</p> <p>vi.02. 1000 cc. T.E. 103½°.</p> <p>25.x.02. 15 cc. T.E. 101°. Several reactions of 100°-101°. Two intravenous T.O. 105 cc. and 1 cc. 17.xi.02. 1 cc. T.O. 102°.</p> <p>12 Jan. 04. T.O. (intravenous) up to 25 cc. Wt. at end 9½ lbs. Well when seen some years later.</p>

* 8½ years later died of phthisis.

5

29. 74. 11½lb.

Sister B. and sister's boy has T.

1 R. L. 41.

2 —

3 "Years."

4 N.

5 V.

6 84-92.

7 / (swelling 100' 2').

8 No Th. Heated.

9 Horn. 1 small.

10 —

11 Treated with T.R. In 13 days temp. N.

12 15.4.02. 25 cc. T.R. 100' 2. Several found 100'.

13 16.02. 1 cc. T.R. 101', wt. 64. 2½lb.

14 17.02. 1½ cc. T.R. 101'. 84. 2½lb.

15 18.02. 2 cc. T.R. 100'.

16 19.02. Wt. 64. 8½lb. Gained 10½lb. and 100 cough.

17 1908. Well. Died in 1908.

6

25. 74. 12½lb.

Brother F. of Addison's disease (T.).

1 R. O. L. 1-41.

2 —

3 Some time.

4 N.

5 V.

6 120-130.

7 / 100', then N.

8 No Th. 101 cc. Old T. 99' 8'; 100' 10'; 100' 2'; 100' 4'; 101 cc., 102'; 101 cc., 103' 4'.

9 [unkn.]

10 Erythema nodosum, and swollen

11

12 Old T. up to 4 cc.; then got 10½lb. 101.02. Quite well. got 2½lb. Remained 101', 102', 103' 4'. First moved to 1 cc.; second course to 2 cc.

5 years after 1910 well.

1908. Well.

7

1908.

74. 10½lb.

Missionary in New Guinea.

1 Stage I.

2 —

3 Remot.

4 N.

5 V.

6 N.

7 / = F. Malaria. Double Tertian.

8 No Th. After treating malaria according to Koch's method, and treating blood of parasite, I tested with isobutylol. 101 cc. Old T. 100' 10', 102'. Again 101 cc., 100' 5'.

9 —

10 —

11 —

12 In the presence of malaria, having was out of the question. I stained first according to Giemsa's modification of Romanowsky's method showed double Tertian infection. Some some beautiful specimens in the sporulation stage showing schromatic zone and chromatic masses brilliancy. Treatment with T.E. up to 2 cc. Remained with several down to 101', 100' 5', 100'. Improved very much. Returned to New Guinea, and two years later died of acute fever with cerebral symptoms (above certain "cerebral" malaria).

1903

8

21. 84. 6½lb.

10.03.

1 Stage I.

2 —

3 More than a year.

4 N.

5 V.

6 N.

7 N.

8 No Th. 1001 cc. Old T. 100' 2'; 100' 10'; 100' 2'; 100' 4'.

9 —

10 —

11 —

12 T.E. up to 5 cc.; few reactions to 100'.

10.03. 2 cc. T.E.; wt. 64. 11½lb.

10.04. Wt. 64. 11½lb.

1908. Quite well.

9

- 11.03. 21. 115.
 Sister. Mother Ep¹ Posttracheo.
 1 R. I-II-III.
 2 —
 3 1 1/2 weeks; lost voice.
 4 A.
 5 B.
 6 N.
 7 N.
 8 No Th. Reacted. 1001 cc. Old T. 1001 cc.; 10015 cc., 10018 cc.
 9 Laryngeal catarrh.
 10
 11
 12 Treated with T.E. to 4 cc.; gained a stage; lost all symptoms; recovered voice.
 1906. Married.
 1908. Five years after treatment, quite well; wt. 140, etc.
 Quite well.

10

35. 60. 135.
 Husband Ep.
 1 R. I-II.
 2 —
 3 Six months.
 4 N.
 5 V.
 6 N.
 7 N. 1001 cc., 1002 cc. Old T. 1001 cc.
 8 No Th. Reaction. Hoarse; cough.
 9
 10 Urinary troubles.
 11
 12 Treated with T.E. right up to 2 cc.; improved greatly in health and looks; wt. 70, etc.
 In 1907 had severe abdominal operation with laparotomy and died next day.

11

- 11.03. 35. 50. 105.
 1 R. I-II-III.
 2 —
 3 Some months.
 4 N.
 5 Moderate.
 6 S.
 7 f.
 8 Th. Reacted. 1001 cc. Old T. 1001 cc.
 9 Ext. cervical glands.
 10

11

- 12 Treated with Old T. up to 12 cc.; lost cough and all symptoms; gained a stage and regained energy.
 Was well 3 years after; lost sight of him since; very poor.

12

- 11.03. 35. 140. 95.
 Brother. 27. 38. 84.
 1 R. I.
 2 —
 3 Less than a year.
 4 A.
 5 B.
 6 N.
 7 N.
 8 No Th. 10015 cc. Old T. 1001 cc.
 9
 10 Spinalgia; nervous chronic strophic rhinitis.
 11
 12 Treated with T.E. to 4 cc.; after course equal to anything; swam 4 miles; reactions 100¹ (7.10 and 12.10).
 11.03. 1005 cc. T.H. 100¹.
 25.10.04. 7 cc. T.R. 100¹.
 In 1908, quite well.

13

- 11.03. 35. 100. 135.
 1 R. I-II.
 2 —
 3 Many years.
 4 A.
 5 B.
 6 N.
 7 N.
 8 No Th. 1001 cc. Old T. 99¹; 1001 cc., 99¹; 1008 cc., 1002¹, 1004¹; reaction lasting 24 hours; ["played out."
 9
 10
 11 a.
 12 Treated with T.E. to 4 cc.; gained 35 lb. and recovered normal energy. Reactions of 100¹ for first two months of treatment; and later 100¹, 102¹, 103¹. After treatment had a very heavy spell of work. He "could not have got through the work before he had treatment."
 In 1908, quite well.

14

1802. 19. 9th. 7th.
 1 I-II.
 2 7.
 3 6 months.
 4 A.
 5 B.
 6 N.
 7 N.
 8 No Th. 1001 cc. Old T. 1000 cc.;
 1013 cc., 1007; 1000 cc., 1011.
 9
 10
 11 A.
 12 Treated with T.E. to 28 cc.
 gained 15 lb.
 Well since.

15

1902. 19. 2d. 6th.
 Sister 4p.
 1 E. I-II.
 2 7.
 3 9 months.
 4 X.
 5 V.
 6 84-92.
 7 N.
 8 No Th. 1001 cc. Old T. 1007;
 1004 cc., 1005; weak and weak.
 9 Done.
 10
 11
 12 Treated with T.E. up to 33 cc.
 gained 15 lb.
 (1002). Wt. 1001. 10th. 1001 rough
 and expectoration.
 Seen late in 1902, quite well.

Central Case.

1902. A.B. 95. Hemorrhage;
 Stage I; physical signs, doubtful.
 No Th. Resisted 1001 cc. Old T.
 1017; went out of hospital appar-
 ently well; refused treatment; re-
 turned in hospital in 1903. Stage
 III; hopeless.

16

1902. 19. 7th. 6th.
 1 I-II.
 2 →
 3 Nearly a year.
 4 X.
 5 V.
 6 N.

7 N.
 8 No Th. 10075 cc. Old T. 987;
 1001 cc., 1007; 1008 cc. T.E.
 [1015].
 9
 10
 11
 12 Treated with T.E.
 15. 10. 1001 cc. T.E. 1015.
 15. 10. 1001 cc. T.E. 995.
 21. 10. 1001 cc. 987.
 20. 10. 1001 cc. T.E. 1002;
 increased rapidly to 2 cc. T.E.;
 as more trouble; then T.E. up to
 1 cc.
 10. 11. 1001 cc. T.E. 994; wt. 50.
 10th. Asthma cured by injection
 though worse after severe doses.
 1908. 10th; no asthma; 9th.

17

1902. 12. 8th. 11th.
 1 E. I-II.
 2
 3 7 years; sympt. of abd. pain, and
 intercostal of Fallopian tubes.
 4 X.
 5 V.
 6 90-110; nervous.
 7 N.
 8 No Th. 1001 cc. Old T. 987; 1002 cc.,
 995; 1004 cc., 1002; and second
 day after dose, 985; 1001, 1002,
 1001, 1007, 1005, 1007, 1004;
 third day, normal.
 9, 10, 11
 12 Treated with T.E. to 2 cc.;
 several reactions of 1007 to 1005;
 5 months after treatment, slight
 reaction; T.E. up to 2 cc.; and
 in 1905, P.T. up to 15 cc.; tested
 May, 1907, Old T. (1015 cc.) nega-
 tive.
 1907. Definite, but no sign of tuber-
 culosis.

18

1902. 12. 8th.
 Sister, mother, and father 4p.
 1 E.O. I. I-II.
 2 →
 3 6 months.
 4 A.
 5 V.
 6 N.
 7 N.
 8 No Th. 1001 cc. Old T. 992;
 1001 cc., 1004.
 9-11 Nil.

- 12 Treated with T.E. up to 4 cc.; gained 3 lbs.; but cough; very severe cramps, with reactions; had to go to bed.
May, 1906, did not react to tuberculin (100 cc. Old T.).
1908. Well.

19

- (c.o.). 25. 7st. 12lbs.
1 R.O. L. L.; also isomorphous signs.
2 —
3 Suffered from "gastritis" and vomiting for 15 months; sent to hospital as "gastric ulcer."
4 N.; 7st. 12lbs.
5 V.; epigastric pain; good appetite, but vomiting all food.
6 76.
7 A.
8 No Th. 100 cc. Old T. 95° F.; 104 cc. Old T. 100° F.
9-11 N.L.
12 Treated with T.E. in 6 months, wt. 9st. 11 lbs.; T.E. up to 2 cc.; gained 27 lbs.
Now her afterwards, was quite well.
Well in 1906; not seen since.

20

- (c.o.). 35. 6st. 9lbs.
Sister recently:
1 R. L. L. I.
2 76.
3 A. post.
4 N.
5 V.; no appetite.

- 6 N.
7 N.
8 No Th. 100 cc. Old T. 100° F.; 100 cc.; 110° F., 99° F., 100° F.; delirious, and severe headache and pains persistent.
9 Intersting acrid-like skin lesion only found in tuberculous subjects (Dr. Murray).
10 Out. Endometritis.
11 A.
12 Treated with T.E. up to 3 cc.; then T.E. up to 1 cc.; then some P.T.O. and finally T.E. up to 5.5 cc.
Nov. 1905. Treated up to 10.5 cc. Old T., and again in April, 1906, up to 104 Old T.; no reaction either time.
May, 1905. Wt. 7st. 12lb.
April, 1906. Wt. 7st. 6 lbs. a gain of 11 lbs.
1908. Well.

21

- 1 Stage I.
2 —
3 Recrudescence.
4 A.
5 L.
6 N.
7 N.
8
9-11
12 Had severe hives, recrudescence gave tuberculosis for a month; hives, ceased, and more than a year later no recurrence, and patient apparently well. Well two years later.

1904

22

- (c.o.). 24. 9st. 12lbs.
1 R. L. L.
2 —
3 1 post.
4 N.
5 V.
6 N.
7 N.
8 No Th. 100 cc. Old T. 99° F.
9
10
11
12 Few doses of T.E., sensitive; then P.T. to 175 cc.; reactions at first, even 105°.

104. 9st. 12lbs.; tested in 1905, 100 cc. Old T. 110°. Continued treatment.
Heard of him in 1907, but have not seen him since 1905.

23

- (c.o.). 5. 2st.
1 Hyaline disease of lung and bronchial tuberculosis.
2
3
4
5
6
7

8 No Th. Hained. 1003 cc. Old T. 101°.

9-11 Little to tell.

12 Case just in as one of pulmonary tuberculosis; deficient movement and absolute dulness at right apex; but bronchial breathing, occasional clicks; treated with tuberculin; P.T.A. up to 90 cc. in two months gained weight; then suspected hydatid disease in right lung; X-rays proved presence of hydatid. Successful operation and now (1908) child is well. Treated in 1906, 100 cc. Old T.; no reaction.

24

6-04 25. Set. 94th.

1 R. I-II.

2 C.

3 Within 4 feet.

4 A.

5 B.

6 N.

7 N.

8 No Th. 1001-1005 cc. Old T. 100°; gen. and local symps.

9

10

11

12 Treated with T.E. in 95 cc.; caught cold; pause for 10 days; then 15 cc. T.E. 103°4'. Cold irritated skin and whole body more sensitive; T.E. again up to 15 cc. v.05. Wt. Set. 12th.; no cough or phlegm, and great gain in energy; 1908. Well.

25

6-04 25. Set. 94th.

Father and mother well. One sister 49.

1 R. I-II, L.I.

2

3 6 months.

4 X; hot rolls.

5 Y.

6 N.; brittle weakness.

7 N.

8 No Th. 10015 cc. Old T. 100°2'; 1005 cc., 100°; severe symps.

9

10 Isomelia; nervous.

11 A.

12 Treated with T.E. up to 5 cc.; till 1.05; pause in June and July. v.05. 9th 94th.; never so heavy before, sleep quite well now.

Reactions:-

11.11.04 102 cc. T.E. 104°6'.

18.11.04 102 cc. T.E. 104°2'.

22.11.04 104 cc. T.E. 100°3'.

24.11.04 106 cc. T.E. 98°4'.

25.12.04 127 cc. T.E. 101°4'.

Sister Wright.

1908. Well.

26

v.04 28. 94.

1 R. I.

2 -

3 3 months.

4 A.

5 B.

6 N.

7 N.

8 No Th. 1001-1005 cc. Old T. 99°8'; bradycardia and vomiting.

9

10

11

12 Treated with T.E. 1000 cc. T.E. 100°4', 1005 cc. T.E. 100°6', T.E. up to 12 cc.; then T.E. and L.I.; returned to T.E.; T.E. up to 15 cc. (100°6'); Set. 11th.

v.05.

2 cc. T.E. 102°4'; wt. 9th 94th. Well since.

27

6-04 22. 94. 94th.

Sister of Mrs. Sharp.

1 I-II.

2 -

3 6 months; treated for "gastric ulcer," vomiting, etc.

4 X. 94. 94th.

5 Y. Vomiting and pain in stomach.

6 94.

7 C.

8 No Th. 100125 cc. Old T. 101°8'; 1001 cc., 101°3'; 1001 cc., 100°4'.

9

10

11

12 Treated with T.E. up to 125 cc.; gained 10th.; 9th 94th.; lost cough and gastric symptoms. Felt well, and being a hospital case, went to a situation in country. Well since, no return of gastric symptoms.

12 Treated with T.E. up to 2 cc.; few reactions in first two months (100', 101', 102' G, 100' G); then up to 4 cc. T.E.

Passed from Dec. to Mar., 1905; then T.E. up to 25 cc. (101'); wt. 84 gms.

Gastric symptoms greatly improved. Case has to be well in December, 1907.

33

1001. 42-70 gms.

1 R. I-II, L. I/2

2 —

3 1 year.

4 X. Lost of wt.

5 Y.

6 80

7 N.

8 No Th. — on Old T. (101')

9 —

10 Dyspepsia; inflame stomach.

11 —

12 Treated with T.E.

12.VI.04. 100g cc. T.E. 100'; later reactions. 100', 100' G; T.E. up to 75 cc.; then T.E. up to 12 cc. 15.VI.04. 84 gms.

18.VI.04. 90 gms.

Fixed pupils; both quite well and able to do anything.

1907. Still well.

34

1004. 20. Oct. 1910.

1 R. I-II.

2 —

3 Six months.

4 X.

5 Y.

6 100-110 (cardiac trouble).

7 —

8 No Th. Treated.

9 Treated with T.E. up to 4 cc.; then P.T.O. up to 5 cc.

Th. improved, but there was also cardiac trouble in this case; I saw patient in 1906.

Well as far as lungs were concerned.

1905.

35

1005. 74. Oct. 1910.

Suffered 40-45 years with her.

1 R. II.

2 —

3 Cough for 18 months.

4 A.

5 R.

6 N.

7 N.

8 No Th. 100g cc. Old T. 100' G; severe symptoms.

9 Hemorrhages.

10 —

12 P.T. up to 75 cc.; severe pain in neck and left limb after doses. Improved greatly and hemorrhages checked; seen late in 1907, then well.

Advised to have more treatment.

1 Y.

2 N.

3 N.

8 No Th. 100g cc. Old T. 101'.

9-11 —

12 Treated with T.E. to 4 cc.

1.VI.05. 81 cc. T.E. 101' G; still ill.

Treated while she was at work; stopped all day long in making skins.

1908 quite well and strong.

37

1007. 25. Oct. 1910.

Status la Stage III, always with her.

1 R. I-II.

2 —

3 Few months.

4 X.

5 R.

6 N.

7 N.

8 Th. in system G. 5; 100; cc. Old T. 102'.

9-11 —

36

1006. 18. Oct. 1910.

1 R. I-II.

2 —

3 Few months.

4 X.

12 Treated with T.E. to 103 cc.; very sensitive. Reactions, several 100' and 101'; still gained wt. but still thin. P.T. up to 35 cc.; wt. 60, 10 lbs. Very great improvement; lost cough, no phlegm. Gained 15 lb. under treatment.
Went back to home in New Zealand.

38

20. 9th 7 lbs.

Orbit doctor same diagnosis.

- 1 R. I-II.
- 2 →
- 3 6 months.
- 4 A.
- 5 E.
- 6 N.
- 7 N.
- 8 No Th. 1001 cc. T.A. 101'; severe in arms and severe symptoms.
- 10 Nervous, felt down very much.
- 12 Treated with T.E. up to 2 cc. Moderate reactions, but across subjective symptoms, especially severe pains.
- 1105 1001 cc. 10 lbs.; no cough or phlegm.
- Gained 15 lbs. under treatment. Married in 1906, and has had a child 1908 well.

39

17. 9th 8 lbs.

Gardener.

- 1 R. I-II; ordered to country.
- 2 →
- 3 Cough for years.
- 4 X. Lost 5 stone.
- 5 Y.
- 6 N.
- 7 N.
- 8 No Th. Resisted.
- 9-11
- 12 P.T. up to 105 cc.; gained 10 lb. Said he felt strong and able to work, and in 1905 was apparently well. 1001 cc. 12 lbs.; gained 10 lb. in two months.
- Heard of as well late in 1907.

40

17. 8th 12 lbs.

Sister in Stage III.

- 1 R. I-II.
- 2 →

- 3 6 months.
- 4 X.
- 5 R.
- 6
- 7
- 8 No Th. 1001 cc. Old T., mistake in recording: 1005-101, 1012'; headache, vomiting, herpes, severe cough.
- 12 Treated with T.E. to 175 cc. Moderate reactions; wt. 1001 lb. In spite of reactions quite well. All Wright's ideas disregarded. Gained 12 lb.
- Well.

41

27. 8th 11 lbs.

Dr. O'N., same diagnosis.

- 1 R. II, L. I.
- 2 →
- 3 Cough when?
- 4 A.
- 5 R.
- 6 N.
- 7 N.
- 8 No Th. 1001 cc. T.A. 101'; shivering, cough, vomiting, pain in limbs.
- 9-11
- 12 Treated by Dr. O'N. with cocaine, cannot vomiting each time, give 11 up. Treated with T.E. 1001 cc. T.R. 101'; 1001 cc. 15 lbs.; T.R. up to 175 cc.; lost cough and phlegm; plenty of energy; never tired now. Pains in same limbs after each dose and knee swollen, also in ankles at times; slipped, and twisted knee, and since then pain after each dose.
- Seen Dec. 1907, quite well.

42

10.05.

- 1 R. I-II.
- 2 (C)
- 3 6 months.
- 4 A.
- 5 E.
- 6 N.
- 7 N.
- 8 No Th. 1001. Old T.: 1005-; wt. 95', 100', 100'2', 100'; symptoms.
- 12 Treated with T.E. to 100 cc.; then T.R. to 6 cc. Dose affected sight, saw rings and blurs.
- Left off treatment on account of private troubles. Active.

43

34- 110, 500.

No family history.

1 R. I. - II.

2 2.

3 6 months.

4

5

6

7

8 1001 cc. Old T. (T.A.) 100' 4' ;

1001, 99' ; 1005, 101 3'.

9

10

11

12 Treated with T.E. up to 4 cc.

Increased energy and full year well.

Well in 1908.

44

21. 500, 400.

1005

1 R. I. - II.

2 2.

3 6 months.

4 N.

5 V.

6 92-100.

7 C.

8 Th. 1 Basted 100 (Old T. (T.A.)

101 3' ; severe symptoms.

9 Pleurisy.

10-11 c.

12 Treated with T.E. ; some severe reactions in hospital ; 102', 102', and afterwards 101', 102', but (not cough) recovered strength and gained 50 lbs. T.E. up to 2 1/2 cc. 1100. wt. 102. 720 ; gained 50 lbs. Have not seen since.

45

8. 30, 300.

1005

Father 3 years ago 80.

1 R. I. - II., and distant one (inter-scapular space thoracic tuberculous).

2 2.

3 5 months ; cough and wheezing.

4 N.

5 V.

6 100-110.

7 N.

8 No Th. 1002 cc. T.A. - 1005 -

1001, 102', 99', 100 1/2.

9 Bronchial tuberculosis.

10 Bronchitic signs.

11 2.

12 Treated with P.T. up to 125 cc. ; vomiting and pain in stomach after doses ; after 102, 103, 104, 1 and 125 cc. reactions 100-102' all cc. 101, 101, 50 lbs. ; also treated with T.A. ; 441 not react to 100 cc. T.A.

13 1907 quite well ; wt. 90, 300 ; gain of 2500, no longer seems due to growth.

46

1005

83. 80, 300.

Shor 80, father and mother well.

1 R. I. - II.

2 2.

3 6 months.

4 N.

5 V.

6 N.

7 C. 99-99 1/2 cc. steps.

8 No Th. 1001 cc. T.A. 99 1/2' ; 1005 cc. T.A. 100 1/2' ; some month earlier and after at night apex ; cough worse.

9 100 lbs.

10 2.

11 T.E. up to 25 cc. ; several severe reactions, 75 cc. T.E. 102', still cc. 1000 to 900 lbs. ; then P.T. up to 325 cc. ; at 100 wt. 100, 100 lbs. ; gained 25 lbs. Had always lived in country (Dulles). 1907 well.

47

1005

70. 60, 300.

1 R. I. - II. ; 1. I.

2 2 well marked signs.

3 2.

4 N.

5 V.

6 N.

7 N.

8 No Th. 1001 cc. T.A. - ; 1005 - ;

101, 99 1/2', 100'.

9

10 Asthma and insomnia from cough.

11 2.

12 Treated with P.T. up to 45 cc. ; gained 10 lbs. ; but feels much better ; sleeps very well now and looks better ; used to cough through the night, now does not cough. 1907. Well.

48

- 18.05. 34. 981 gms.
 1 R. L.-H., also scattered foci of pleurisy.
 2 —
 3 1 to 3 months.
 4 A.
 5 B.
 6 —
 7 —
 8 No Th. 10005 cc. T.A.—: 10018—: 1007, 1017 for three hours; severe sympts.
 9 Flushing over half right lobe.
 10 —
 11 —
 12 Began with 1004, very severe reaction.
 2.viii. 1 A.M. 1015.
 3.viii. 2 P.M. 1015.
 4.viii. 5 P.M. 1015.
 Then treated with T.R. up to 2 cc. wt. 1101, 5 lbs.; hot pain and cough Jan. 1908; well; severe reactions did not recur; gained 25 lbs.
 1908. Well.

49

- 18.05. 35. 561 gms.
 No family history.
 1 R. L.-H.
 2 —
 3 Pleurisy in 1900.
 4 X; discoloration of skin.
 5 Y; anemia.
 6 120.
 7 N.
 8 No Th. 1000 cc. T.A. 997'; 1001, 1004'.
 9 —
 10 Splenic anemia, enl. spleen (Bast's disease).
 11 —
 12 Treated with T.E.
 7.viii.05. 1007 cc. T.E. 101'.
 12.viii.05. Began with T.E.
 14.viii.05. 1005 cc. T.E. 101'.
 14.viii.05. 91 cc. T.E. 1002'.
 19.viii.05. 75 cc. T.E. 100'.
 25.viii.05. 2 cc. T.E. 100'.
 2.xi.05. 25 cc. T.E. 101'4' wt. 561, 5 lbs.
 18.xi.05. 9 cc. T.E. 101'.
 1.xii.05. Used P.T. up to 125 cc.; wt. 581, 5 lbs.
 Became pregnant during treatment, but aborted; treated by local physician.

Died in vi. 1907 of Bast's Disease or splenic anemia.

50

- 18.05. 37. 561 gms.
 No family history.
 1 R. L.-H.
 2 Sympt. of anemia.
 3 For some months.
 4 X.
 5 Y.
 6 120.
 7 N.
 8 No Th. 1000 cc. T.A.; said to be 110'; more likely 100'4'.
 9 Repeated. 1000 cc. T.A. 101'4'.
 10 Anemia and bronchitis rules.
 11 6.
 12 Treated with T.E.
 21.vi.05. 1 cc. T.E. 100'.
 22.vi.05. 5 cc. T.E. 100'.
 29.vi.05. 55, 100'.
 5.vii.05. 55, 100'.
 12.vii.05. 100 P.T. 101'.
 17.vii.05. 1025 P.T. 99'6'.
 1.viii.05. 2 P.T. 101'.
 10.viii.05. 2 P.T. 99'9'.
 16.viii.05. 25 P.T. 100'4'.
 23.viii.05. 61 P.T. 101'2'.
 2.viii.05. 2 P.T. 100'.
 7.ix.05. 1 P.T. 100'.
 12.viii.05. 15 P.T. 99'2'.
 21.viii.05. 4 P.T. 99'.
 27.viii.05. 5 P.T. 99'.
 20.ix.05. 75 P.T. 98'4'; wt. 604, 4 lbs.
 Silent Weight.
 1907. Treated, negative.
 1908. Well.

51

- 1 Stage 1.
 2 —
 3 4 months ago: Flushing.
 4 Thin.
 5 B.
 6 N.
 7 N.
 8 No Th. 1000 cc. T.A. 1002'.
 9 11.
 10 Treated with P.T. up to 55 cc.; daily dose caused temp. of 101'2', otherwise no trouble; hot cough and felt well.
 Believe him to be well, though I have not seen him.

52

1005 2d. 1st. ghs.

- 1 Stage I-II
2 —
3 I
4 X; 1st. ghs.
5 V
6 N
7 N
8 No Th. 1001 cc. T.A. 1004';
1002, 1003.

- 9-11
12 Treated with P.T. up to 105 cc.;
then T.E. up to 4 cc.
1006. "Can walk splendidly and
"do anything now—could not
"walk when I came." Wt.
7th. 2 lbs.; gained 12 lbs.
Soon since, quite well.

53

1006 17. 1st. ghs.

- 1 Stage I.
2 —
3 Influenza 7 five months ago, temp.
100—102'; for five weeks cough,
phlegm, croup, short of breath.
4 A.
5 B.
6 N.
7 N.
8 No Th. 1001 cc. Old T. 993';
1002 cc. Old T. 995'; 1005 cc.
Old T. 101'.

- 9
10 Fits of coughing, due to enlarged
lingual tonsil.
11 A.
12 Previous treatment: for three
months treated for cough and
croup, and patient got gradually
worse till she came to me and
Tuberculin was used.
Tuberculin treatment: treated with
P.T. up to 105 cc.; she gained wt.,
recovered health and energy and
has no cough nor phlegm; wt. 9 lb.
Soon is 10 lb. quite well.

54

1007 22. 1st. ghs.

- 1 Stage I.
2 —
3 Cough for 2 years.
4 X.
5 V; indigestion.

- 6 N.
7 N.
8 No Th. 1001 cc. T.A. 1003';
1002, 1003, 1004.
9-11 Nil.
12 Treated with P.T. up to 105 cc.;
1002, 1003.
13 10 lb. 1006, 1007, 1008. Never felt
so well in her life; her cough, and
the indigestion.
1008. Treated up to 105 cc. T.A.—

54a

1008 24. 5th.

- Brother 4 lb (galloping 4 lb.
1 I. I-II.
2 —
3 Recrud.
4 X.
5 B.
6 N.
7 (1002').
8 No Th. 1001 cc. T.A. 1004'.

- 9
10
11
12 Treated with T.E. up to 105 cc.;
gained ghs. and lost cough.

55

1009 25. 7th. ghs.

- No family history.
1 Stage I.
2 —
3 6 months to a year.
4 X.
5 V.
6 N.
7 N.
8 No Th. 1001 cc. T.A. 992';
1002, 993'; 1005, 1004'.
9 Hemorrhage, small for some days.
10-11
12 Treated with P.T. up to 7 cc.;
gained ghs.; fever so heavy is
1 lb.; looked very well; no cough.
Treated 1006 up to 105 cc.—
1008. Well.

56

1010 26. 7th. 1 lb.

- Bad history in cousins.
1 I-II.
2 —
3 2 years.

- 4 X.
5 V.
6 So—gl.
7 N.
8 No Tn. 100 cc. T.A. 101'.
9
10 Neuritic: injections very painful.
11
12 Treated with T.E. up to 103 cc.:
injections, 1000', 1000', 1000', 1000',
100', 1010'; all, wt. 84.5 lbs.;
then T.E. up to 12 cc.; lost 3 lb
also; then few doses of P.T.O.
(12 cc.) and T.E. (25 cc.) wt.
84.5 lbs.; then P.T.
Log. P.T.O. up to 75 cc.; then
P.T. up to 4 cc.
100. Sat. 12 lbs.; "now so heavy
"before"; gained 12 lbs.
1000. Well.

57

11.05. 22. 94. 94.5
Sister Ep.

- 1 R. I.-II., L. II.
2 —"
3 2 years.
4 X.
5 II.
6
7
8 Tn. G. 1-2
9 Hemorrhages.
10-11
12 Treated with P.T. up to 8 cc.;
wt. 15 lbs.; many reactions, 100',
101', 102'.
10.11.05. 5 P.T. 102'4'.
X.100. 5 P.T. 100'.
15.1.06. 55 P.T. 100'; wt.
94. 13 lbs.; great improvement in
health and physical signs; no
cough and no phlegm; gained
wt.; few sputa now; before
cracking and bubbling riles.
Not over size.

58

11.05. 15. 100.

Brother in Stage II.-III.

- 1 Stage I. (R. I.-II.).
2 —"
3 Few months.

- 4 A.
5 R.
6 N.
7 N.
8 No Tn. 100 cc. T.A. 101';
very severe in arm and temple.
9
10-11
12 Treated with P.T., sensitive.
2.10.05. 1000 P.T. 102'4'; patient
doubtless does also 1000 cc. P.T.
1000'; 1000 cc. P.T. 100'4';
then P.T.O. up to 55 cc.
Apparently well, but have advised
more treatment if test be positive.

59

11.05. 21. 120. 120.
Mother Ep.

- 1 Stage I; pronounced bluish by
last doctors; passed for A.M.P.
Society and passed by railway
doctor.
2 —"
3 Short of breath for 6 months and
loss of energy.
4 A.
5 V.
6 N.
7 N.
8 No Tn. 100 cc. T.A. 102'2'.
9-10
11 A.
12 Treated with P.T.; shot, a few
smaller doses.
3.11.05. 1000 cc. P.T. 100'.
10.11.05. 1005 cc. P.T. 100'.
15.1.06. 1007 cc. P.T. 99'.
20.3.06. 1005 cc. P.T. 98'.
2.12.05. 75 cc. P.T. 100'; no
further reaction above 100'; P.T.
continued up to 75; patient was
tested in May, 1907, wt. 124.
13 lbs., and did not react to
1005 cc. T.A.; he has regained
all his energy; can do anything
and is very strong; he had hard
work in the railways and con-
tinued at work throughout treat-
ment *de jure* of reaction; I
arranged larger doses so that
reactions occurred when he was
off duty.

1906

60

18.06.

No family history.

1 Stage I.

2

3 Cough 3 years; phlegm 2 years.

4 X.

5 B.

6 N.

7 N.

8 No Ph. Record.

9-11

12 P.T.O. few times; then treated with P.T. up to 75 cc.; wt. 120 lbs.

Felt quite well, had no return in country.

61

18.06.

25. 40. 4 lbs.

History up vii, vii.

1 R. I.-II; sticking ribs.

2 ———

3 Cough for 14 days.

4 Lost a stone.

5 B.

6

7

8 No Th. Record.

9-11

12 Treated with P.T.O. to 1 cc. with sections; then T.O.A. up to 975 cc.; then P.T.O. up to 1 cc.; 90. 18 lbs.; then P.T. up to 50 cc. (18.06); weight 107 up to 935 cc. T.O.A. (20.11); wt. 100; gained 10 lbs., and felt and looks well.

62

18.06.

24. 70. 15 lbs.

Pregnant.

1 R. I.-II.

2

3 Recent.

4 X.

5 V.

6

7

8 No Th. 100 cc. T.O.A. 1007, 99 5/8; 1006, 100 5/8, 100 5/8.

9

10-11

12 Treated with P.T.O. up to 1 cc.; then P.T. up to 7 cc.; gained weight and strength.

63

18.06.

25. 40. 8 lbs.

Mother wt. 54. 40.

1 R. I.-II.

2 ———

3

4 A lot of strength.

5 B.

6 N.

7 N.

8 No Th. 1002 cc. T.O.A. 1007; phlegm, half an ounce, yellow.

9-11

12 Treated with P.T.O. up to 1 cc.; then P.T. to 75 cc.; 1 inst. 6 lbs.; had to go off to shooting; felt well and strong, gained 10 lbs.

64

18.06.

24. 80. 5 lbs.

Spent much time with boy who died of Ph.

1 R. I.-II. Pneumy and ribs.

2 ———

3 Recent.

4 A.

5 B.

6 N.

7 N.

8 No Th. 1003 cc. T.O.A. 1007; swollen arm, bowelache, nausea, sleep.

9-11

12 Treated with P.T. up to 75 cc.; 80. 18 lbs.; gained 4 lbs.; and felt quite well; no physical signs.

65

18.06.

33. 80. 18 lbs.

1 Stage I.-II.

2 ———

3 Six months. Pneumonia 6 months ago, cough ever since, much blood in expectoration.

4 X.

5 V.

6 N.

7 N.

8 No Th. 91 cc. T.O.A.—; 90—; T.O.A. 107.

9

10 Ph. is 18.06; ill 6 weeks, cough ever since.

11 C.

- 12 Treated with P.T.O. up to 125 cc.; then T.O.A. up to 3 gr.; reactions 101° (1025 cc.); 100° 1/2 (125); then P.T. up to 75 cc.
Last cough, looks very well, infinitely better, and feels quite well. Went back to New Zealand.

66

18.06. 32. 5th. 5th.
Name.

Advised by doctor to see me. Brother 4p and sister 4p.

1 R. I-II.

2 →

3 Cough and hoarseness for 2 years.

4 N. Lost a stone in 10 months.

5 Y.

6 N.

7 N.

8 No Th. 1001 cc. T.A. 106°; arm swollen, cough and hoarseness.

9

10

11 4.

12 Treated with P.T.O. to 7 cc. In 3 months no cough, and wt. 8st. 12½lb.; walking all the time; afterwards P.T. up to 775 cc.; wt. 9st. 12½lb.; tested 9 months later: 1005, 1015, 101, 1045 cc. T.A. all negative.
Stage quite well and looks very well.

67

32. 1st. 4th.
Sister 8, also husband has 9.

1 R. I-II.

2 →

3 Cough for 18 months.

4 A.

5 Y.

6 N.

7 N.

8 No Th. 1001 cc. T.A. 101° 1/4; very severe in arm.

9-11 N.

12 Treated with P.T.O.

15.viii.06. 1001 P.T.O. 1008°.

21.viii.06. 1005 P.T.O. 1012°.

29.viii.06. 10015 P.T.O. 101°; then T.R.

5.viii.06. 10000 T.R. 99°.

11.viii.06. 10005 T.R. 100° 1/2.

24.ix.06. 1015 cc. T.R. 102°.

2.x.06. 1015 cc. T.R. 99° 1/2.

7.x.06. 98 cc. T.R. 99°.

10.x.06. 101 cc. T.R. 98°.

12.xi.06. 172 cc. T.R. 98°.

19.xii.06. 145 T.R.

27.xi.06. 179 cc. T.R.

Sister's death interrupted treatment.
Walterer decr.—Silent Wright.

68

viii.06. 33. 8th. 5th.

Elderly sister 4p.

1 R. I-II.

2 →

3 Very liable to catch colds.

4 A.

5 B.

6 N.

7 N.

8 No Th. 1001 cc. T.A. 100° 1/2.

9 Catarrh.

10 Frequent colds.

11

12 Treated with P.T.O.

5.viii.06. 1001 cc. P.T.O. 101°.

9.viii. 100°, 102°.

15.viii.06. 1001 cc. P.T.O. 101°.

21.viii.06. 1001 cc. P.T.O. 99° 1/2;

no further reactions (Silent Wright).

P.T.O. up to 75 cc.; then T.O.A.

up to 1 cc.; then P.T.

20.xi.06. 1775 cc. P.T. 100° 1/2.

27.xi.06. 1225 cc. P.T. 98° 1/2.

(Silent Wright) up to 505 cc.

P.T. 98° 1/2.

viii.06. Tested, no Th. T. 100° 1/2.

Further course with T.R. up to 2 1/2

cc.; no reactions; wt. 8st. 7½lb.

Dec. 1907. Very well.

69

viii.06. 37. 8th. 12th.

Mother in Stage III.

1 Stage I.

2 →

3 Recent (3 months).

4 A.

5 Y.

6 N.

7 N.

8 No Th. 1001 cc. T.A. —; 10005—

1005, 1008°, 101°, 101° 1/2, 101°.

98°, 97° 1/2.

9-11 N.

12 Treated with P.T.O. up to 1 cc.;

then T.O.A. 3 cc.; then P.T.

up to 75 cc.; lost cough, no

phlegm, appetite good; feels quite

well. 1907.

70

11.11.06. 16. 7st 8lbs.
Father 4p is 1000; also half-brother
and sister 4p. Other father has 4p.

- 1 R. I.-II. Well marked.
- 2 —→
- 3 6 months.
- 4 X.
- 5 V.
- 6 N.
- 7 N.
- 8 No Th. 1000 cc. T.A. 100'; 1000,
400'.

9-11 Nil.
12 Treated with P.T.O.
13.11.06. 1005 cc. P.T.O. 100'.
15.11.06. 1007 cc. P.T.O. 100' 2'.
18.11.06. 1005 cc. P.T.O. 99'.
20.11.06. 1002 cc. P.T.O. 99' 2'.
23.11.06. 1003 cc. P.T.O. 99'.
26.11.06. 1005 cc. P.T.O. 99' 4'.
29.11.06. 91 cc. P.T.O. 103' 2'.
31.11.06. 101 cc. P.T.O. 98'.
30.12.06. 102 cc. P.T.O. 98'; no
further reaction.
31.12.06. 105 cc. P.T.O. 100'.
6.1.07. 1 cc. P.T.O. 98' 4'; then
P.T.
17.1.07. 61 cc. P.T. 100'.
22.1.07. 104 cc. P.T. 99'.
24.1.07. 125 cc. P.T. 101'.
26.1.07. 125 cc. P.T. 99' 2' and
afterwards no more reactions.
5.2.07. 7.11. P.T. 98' 4'.
Treated 2.07. 1005, 1015, 1015, 104;
throughout patient was at work all
day long from 9 till 5, except for
first 2 months of treatment; not a
day out of Sydney.
1908. Well.

71

1.06. 26. 9st 8lbs.
Brother, 1st. 40. 4p.

- 1 R. I.-II., L. I.
- 2 —→
- 3 7 months ill.
- 4 Usually 5 st; after rest, 9st 11lbs.
- 5 R.
- 6 N.
- 7 N.
- 8 No Th. 1000 cc. T.O.A.—1025 T.O.A.,
99' 4'; 101 T.O.A., 100' 2'; severe
reaction in arm.
- 12 Treated with P.T.O.; very sensi-
tive.
20.1.06. 1005 cc. P.T.O. 100' 4'.
3.2.06. 10005 cc. P.T.O. 100' 8'.

8.2.06. 10005 cc. P.T.O. 99'.
11.2.06. 1003 cc. P.T.O. 99' 8'.
14.2.06. 1005 cc. P.T.O. 101' 6';
then T.R.
20.2.06. 1002 T.R. 100' 2'.
25.2.07. 2 cc. T.R. 100' 5'.
4.3.07. 25 cc. T.R. 100' 2'; no
reaction ill.
10.3.07. 174 cc. T.R. 101' 3'.
18.3.07. 173 cc. T.R. 98' 4'.
18.3.07. 2 cc. T.R. 99' 8'.
Felt well.
Well late in 1907.

72

11.11.06. 27.
Mrs. G.

Husband in second stage 4p.
1 R. I.-II.
2 —→
3 Few months.
4 X.
5 V.
6 N.
7 1/2. 99' 4'; 99' 6'; E.
8 No Th. Kneaded; 1001 cc. T.A.
100'.
9-11 1/2.
12 Treated with P.T.O.
1.12.06. 1000 cc. P.T.O. 100' 6'.
5.12.06. 1002 cc. P.T.O. 99'.
12.12.06. 1003 cc. P.T.O. 100'.
21.12.06. 102 cc. P.T.O. 100'.
23.12.06. 105 cc. P.T.O. 99' W
then T.R.
18.1.07. 2 T.R. 99' 4' up to 185
cc. 100'.
1908. Apparently well, but should be
sewed.

73

21.06. 32. 10st 6lbs.

- 1 R. I.-II., R. I.
- 2 —→
- 3 Since Christmas 1903 (4 months),
has been ill.
- 4 X.
- 5 R.
- 6 N.
- 7 N.
- 8 No Th. 1000 cc. T.A. 100' 5'.
9 Hemorrhages moderate.
10 Influenza? and sweating.
11 1/2-cc.
12 Treated with P.T.O. up to 19 cc.;
T.O.A. up to 15 cc.; wt. 11st.
4lbs.; then P.T. up to 125 cc.

1.vi.06. T.E. up to 1 cc.; wt. 1107.
70 lb. Pulse.

Then:

- 1.ii.06. 775 P.T. 100°2'.
5.ii.06. 2 cc. P.T. 100°.
10.ii.06. 25 cc. P.T. 100°2'.
25.ii.06. 25 cc. P.T. 100°2'.
30.ii.06. 1 cc. P.T. 99°5'.
7.iii.06. 15 cc. P.T. 99°5'.
12.iii.06. 2 cc. P.T. 101°. Pulse.
Then P.T. again up to 25 cc.;
wt. 1181 34 lbs.; later, 1211 37 lbs.;
gained 21 lbs.; working as tram
conductor all the time; now looks
and feels well.

74

1.vi.06. 15. 50. 4 lbs.

Clinical Ep.

- 1 R. II, L. L-II.
2 —
3 1 year.
4 X.
5 R.
6 N.
7 C.
8 Th. G. J. 50 spines.
9-10-11. Nil.
12 Treated with P.T.O.; gained 7 lbs.
12.ii.06. 85 cc. P.T.O.
So much improved in every way that
he did not think he needed any
more treatment. Have not seen
him since.

75

1.vi.06. 8. 20. 15 lbs.
Hospital case.

- 1 Tuberculous adenitis; enlarged
glands in both submandib. regions,
esp. right.
2 —
3 1 month.
4 A. 21. 13 lbs.
5 R.
6 72.
7 C.
8 No Th. 100025 Old T. 101°; typi-
cal reaction in arm and in right
knee, some pain.
9 Tuberculous disease of knee a year
ago.
10
11
12 Treated with P.T.O. up to 125 cc.;
wt. 30. 64 lbs.; then P.T. up to
150 cc.; wt. 36. 64 lbs.
Glands much smaller; knee feels
quite well; great improvement.

76

21.06. 15. 100. 12 lbs.

Solar Ep.

- 1 Stage L-II.
2 —
3 1 month.
4 A.
5 R.
6 N.
7 N.
8 No Th. 101 cc. T.O.A. 102°.
9-11
12 Treated P.T.O. but reacted; then
treated with T.R. At first lost
weight and was very pleased;
down to 100. 4 lbs., but gained
again and went steadily forward.
25.ii.06. 100 cc. P.T.O. 101°; then
T.R.
2.iii.06. 100 T.R. 101°.
10.iii.06. 100 cc. T.R. 95°4'.
1.ii.06. 100 cc. T.R. 99°.
21.ii.06. 2 cc. T.R. on to 2 1/2 cc.
T.R.
In February (1907), few reactions of
100°; looked and felt well; 111.
5 lbs.; gained 7 lbs.

77

v.06. 30. 100. 12 lbs.

P.A. Hospital.

- 1 Pain and swelling with much effu-
sion in right ankle; movements
limited; of pain; pain also in
subtalar joints; diagnosed as
gonorrheal rheumatism, and
treated as such by Dr. M. Then
Dr. M. asked me to treat patient.
2 —
3 10 months.
4 Pale, fatty.
5 R.
6 70.
7 N.
8 No Th. 101 cc. Old T. 100°4';
100125, 99°5'; 1004, 105°.
1 Frictional stress leading to prostatic
abscess (1911).
10-11
12 Treated with P.T.O.
21.vi.06. 100 cc. P.T.O. 100°.
30.vi.06. 51 cc. P.T.O. 92°5'.
7.vii.06. 100 cc. P.T.O. 99°2'; wt.
121. 64 lbs.
26.vii.06. 100 cc. P.T.O. 105°.
4.viii.06. 105 cc. P.T.O. 101°.
21.viii.06. 1 cc. P.T.O. 95°4'.

30.11.06. '4 cc. P.T.O. 98'4'.
 3.12.06. '1 cc. P.T.O. 101'4'.
 10.12.06. '35 cc. P.T.O. 100'.
 15.12.06. '4 cc. P.T.O. 99'.
 24.12.06. '5 cc. P.T.O. 98'4'.
 3.1.07. '5 cc. P.T.O. 100'; 1341.
 1015. Feels well; no pain; swelling
 leg much diminished and almost
 healed; gained 25 lbs.

78

vi.06. 30. 841. 1115.

Brother and sister have p.

1 R. II., L. I-II; sinusitis - 1115.
 2 —
 3 4 years ago, hemorrhage, loss 2
 quarts.
 4 N.
 5 Y.
 6 N.
 7 N.
 8 No Th. Resisted.
 9 Hem. 2 years ago and 2 year and
 5 months ago.

10
 11
 12 Treated with P.T.O.
 23.11.06. 100% cc. P.T.O. 100'8'.
 3.12.06. 100% cc. P.T.O. 98'4'.
 10.12.06. 101 cc. P.T.O. 98'4'.
 14.12.06. 102 cc. P.T.O. 98'4'.
 21.12.06. 100 cc. P.T.O. 102'6'.
 28.12.06. 100 cc. P.T.O. 98'4'.
 29.12.06. 95 cc. P.T.O. 101'4' wt.
 101. 1115.; gained 1115.
 28.1.07. 105 cc. P.T.O. 100'.
 27.12.06. 101 cc. P.T.O. 98'4'; then
 P.T. after 3 doses.
 5.1.07. 105 cc. P.T. 101'.
 12.1.07. 105 cc. P.T. 101'.
 22.1.07. 105 P.T. 101'4'; advised
 pause.
 10.2.07. Wt. 901. 1115.; gained
 1115. and lost cough; and looks
 picture of health. Another course
 up to 100%; wt. 1001. 1115.; all
 signs have disappeared.
 1.3.07. P.T.O. up to 100 cc.; then
 P.T. up to 125 cc.; wt. 901. 1115.
 never more than 341. 1115. before
 illness; gained 1115.; at one
 time 1115.
 1908. Well.

79

vi.06. 30. 801. 1115.

Stage 1.

1 Stage I.

2 —

3 Few weeks.

4 X.

5 Y.

6 N.

7 N.

8 No Th. Resisted.

9 —

12 Saw Dr. S. S., who said right lung
 was affected, and told her to spend
 a year in the country and sleep on
 balcony. She did neither, but had
 tuberculosis, with result appended.

Treated with P.T.O. to 100 cc.; gained
 1115.; then T.O.A. 1 cc.; wt.
 1001. 1115.; then P.T. to 105 cc.;
 wt. 1001. 1115.; gained 2115.

80

vi.06. 30. 901. 1115.

1 Stage I.

2 —

3 —

4 A.

5 Y.; indigestion; loss chiefly in
 vegetables.

6 N.

7 1. 99', 99'4'.

8 No Th. 1001 cc. T.A. 99'2';
 1005, 99'2'; 91 cc. 100', 101', 102'.

9 Hemorrhage 2 years ago 341.

10 Indigestion.

11 A.

12 Treated with P.T.O. up to 101 cc.;
 T.O.A. up to 101 cc.; then P.T. up
 to 105 cc. (2.12.06).

13.12.06. 105 cc. Old T. 101'6'.

25.12.06. 105 cc. P.T. 101'2'; at
 101'6' on 2.12.06 105 cc. P.T. and
 not only temperature at all; wt.
 901. 1115.

Temperature gradually fell to normal,
 and now patient can lie on right
 side; could not do so before treat-
 ment.

81

vi.06. 30. 1111. 1115.

Wife in Stage III., since dead.

1 Stage I.

2 —

3 12' for 3 months.

- 4 A.
5 B.
6 N.
7 N.
8 No Th. 904 cc. T.A. 98'2';
'005, 99'2'; '006, 101'3'. Herpes,
headache, rough skin.
9-11
12 Treated with P.T.O. up to 1 cc.;
then P.T. to 65 cc.; no reactions
of any degree; wt. 11st. 8½lb.;
gained 7½lb.; lost cough and looks
well.

Advised testing, but he thought it
unnecessary.

Control Case.

- L. N. 21. Tested in 1902 by
me, in P.A. Hospital and re-
sulted; Stage I., physical signs
slight, would not have treatment;
came to see me privately and told
his own history exactly; was in
Stage III. four years later, hyper-
tens.
1906.

1907

82

- 1.07. 21. 7st. 4½lb.
No family history.
1 Stage I.-II. (B.).
2 —
3 Since Christmas 1906.
4 X; lost few pounds.
5 B.
6 N.
7 N.
8 No Th. 101 cc. T.A.—; '002, 100';
'005, 102'.
9
10 Very severe pains after doses; in-
side from hip down; had this pain
before doses; "pains in chest and
back, and in all joints, fingers, and
thumbs" after dose.
11 A.
12 Treated with P.T.O. up to 75 cc.;
gained ½lb.; then P.T. up to
'075 cc.; no reaction; gained 6½lb.
In 1908, tested and did not react.
1908. Feels quite well, and able to
do everything.
Tested 1.10.8. No reaction to 94 cc.
Old T.; wt. 7st. 12½lb. Feels
splendid.

83

- 1.07. 21. 9st. 6½lb.
Brother under treatment also.
1 Stage.
2 —
3 Recent.
4 A.
5 B.
6 N.
7 X. 100'—100'6'.

- 8 No Th. 1001 cc. T.A. 99'5';
'000, 99'8'; '005, 100'5'. For 24
hours.
9-11
12 Treated with P.T.O. up to 8 cc.;
wt. 9st. 12½lb.; then P.T. up to
'95 cc., 100. 0½lb. Improved in
every way; temperature gradually
fell to normal.
1908. Well.

84

- 1.07. 17. 7st. 1½lb.
Uncle had haem. of lung.
1 R. I.-II.; and enlarged glands in
neck.
2 —
3 Few months.
4 A.
5 B.; app. good.
6 N.
7 N.
8 No Th. 1001 cc. T.A. 105', last-
ing 24 hours.
9 Enlarged cervical glands.
10-11
12 Treated with P.T.O.
22.1.07. 1005 P.T.O. 101'2'; cough,
swelling and pain in glands of
neck, lasting two days; headache
and vomiting.
23.1.07. 1007 cc. P.T.O. 101'.
24.1.07. 10025 cc. P.T.O. 102'.
14.2.07. 1004 cc. P.T.O. 100' up
to '75 cc.; then P.T.
27.2.07. 102 cc. P.T. 101'2'.
5.3.07. 1015 cc. P.T. 99'.
20.3.07. 102 cc. P.T. 95.4' up to
'55 cc.
Quite well.

85

807. 30. 70. 1114.

Father etc. 35 & acute pleurisy.

1 R. H. L. 1-II; many ribs.

2 —

3 2 or 3 months ago.

4 N.

5 Y.

6 N.

7 N.

8 No Th.

9-11

12 Treated with P.T.O. up to 3 cc.;
 Sat.; but lost 4th. after Alexander
 operation, etc., while under treat-
 ment; then P.T. up to 3 cc.; sat.
 Sat. 6th. on to 7th cc.; was treated
 for some months at a distance;
 does not regularly and infected
 by sister. Wrote to say she felt
 quite well; never better in her
 life; sat. cough, no spasm, able
 to do anything now.

1908. Well.

86

807. 30. 90. 480.

1 R. 1-II.

2 —

3 Air. 5. Pleurisy.

4 A.

5 R.

6 N.

7 N.

8 No Th. 1000 cc. Old T. 1005-1010.

100.5; 101, 102.6.

9

10 Arthros.

11 A.

12 Treated with P.T.O. up to 4 cc.;
 then P.T. up to 3 cc.; sat. 54th.
 ended 6 months after 1005, 1015,
 1025, 1045 cc. T.A. Can now
 throw off colds, feels and looks
 well.

1908. Well.

87

807. 25. 74. 414.

1 Stage I.

2 —

3 Small hemorrhage in 21 cc.

4 N; lost weight.

5 R.

6 100-114; pulse variable, also
 systolic murmur at apex.

7 N.

8 No Th. 1000 cc. Old T. 1005-1010;
 101, 102.6; for 24 hours, and still
 weak, never better.

10 Treated with P.T.O. up to 1 cc.
 1005 P.T.O. 101; in a month
 "felt greatly" "began to feel so
 well"; sat. 124th; then P.T.
 up to 75 cc.; "aching in back,"
 rather worse after done.

88

807. 27. 75. 714.

1 Stage I-II.

2 —

3 Severe hemorrhage 3 rows ago;
 colour in sputum since.

4 A.

5 R.

6 N.

7 N.

8 No Th. 1000 cc. T.A. 1012;
 lasting 24 hours.

9-11

12 Treated with P.T.O. to 1 cc.;
 and then P.T. to 3 cc.

89

807. 2. 100. 844.

Brother and sister had 9.

1 R. 1-II.

2 —

3

4

5

6

7

8 No Th. 1000 cc. T.A. 101;
 marked symptoms.

9-11

12 Treated with P.T.O. to 75 cc.;
 then P.T. up to 1 cc.; 1002, 1014.
 Looks well, no cough now, before
 troublesome; says he feels quite
 well and strong.

90

807. 18. 101. 644.

Four brothers and one sister 8p.

1 Stage I-II.

2 —

3 Cough for fortnight.

4 A.

5 R.

6 N.

- 7 N.
8 No Th. '003 cc. T.A. '100'; very great swelling in arm, edema, colic; also headache, loss of appetite, vomiting.
9-11
12 Treated with P.T.O. to 1 cc.; wt. 1001.
12.vi.07. '003 cc. P.T.O. '101'.
21.vi.07. '001 cc. P.T.O. '000'.
25.vi.07. '006 cc. P.T.O. '100'.
30.vi.07. '0075 cc. P.T.O. '094'.
1.vii.07. '01 cc. P.T.O. '000'.
10.vii.07. '01 cc. P.T.O. '1004'.
25.vii.07. '04 cc. P.T.O. '094'.
22.viii.07. '085 cc. P.T.O. '084'; no more reactions on 10 cc.; then P.T. up to '95 cc.; wt. 1006. Great improvement; gained 7 lbs.

91 ✓

- vi.07. 17. Oct. 1916.
Sister 26 this year, and mother 26 2 years ago.
1 Stage I-II.
2 —
3 Cough for a month.
4 A.
5 B.
6 N.
7 N.
8 No Th. '001 cc. T.A. '1003'.
9-11
12 Treated with P.T.O.; very sensitive.
18.vi.07. '0075 cc. P.T.O. '101'.
20.vi.07. '0065 cc. P.T.O. '084'.
21.vi.07. '0075 cc. P.T.O. '084'.
27.vi.07. '0075 cc. P.T.O. '101'.
5.vii.07. '0125 cc. P.T.O. '101'.
15.vii.07. '01 cc. P.T.O. '100'.
23.viii.07. '00 cc. P.T.O. '084'; no further reactions up to '9 cc. P.T.O.; wt. 921. 7 lbs.; then P.T. up to '05 cc.
Felt as well that he did not think he needed more treatment.

92 ✓

- iv.07.
Mother in Stage II. Th. G. L.
1 Stage I.
2 —
3 Recrud.
4 X. Anemic and listless.
5 V.

- 6 N.
7 N.
8 No Th. '00075 cc. T.A. '100', '100', '1004'; arm swollen, red and tender.
9-11
12 Treated with P.T. up to '2 cc. Has now healthy child, full of energy, never tired; apparently in best of health.
Should be tested.

93 ✓

- iv.07. 29. Oct. 1916.
No family history. Public school teacher.
1 Stage I.
2 —
3 Cough for 2 years.
4 A.
5 B.
6 N.
7 F. '094'.
8 No Th. '002 cc. T.A. '102', '1024'; '100', '094', '1014', '104'; '014', '094'; '084', '094'; severe headache; rough voice; severe local reaction in arm.
9-11
12 Treated with P.T.O. up to 1 cc.; then P.T. up to '725 cc.; 21st. 5 lbs.
Tested in Dec. 1907. '003 cc. T.A.—

94 ✓

- iv.07. 24. Oct. 1916.
Sister has had only glands in neck, and another reacted to tuberculin.
1 Stage I-II.
2 —
3 Cough for 4 months.
4 Tall and thin; X.
5 Y.
6 N.
7 N.
8 No Th.
9-11
12 Treated with P.T.O. up to 1 cc.; wt. 921. 3 lbs., gain of 6 lbs.; then P.T. up to '6 cc. Had much worry and trouble, and lost wt. 800. 5 lbs.; but felt well and less cough altogether.
Should be tested.

- 9
10-11
12 Treated with P.T.O. up to 1 cc.; then P.T. up to 50 cc.; gained 15 lbs.; wt. 50t. lbs.
Very well; no signs of active disease; no cough.

100

- 11.07. 23. 300t. 48lb.
Brother 46, saw him in Stage III., sent him home.
1 Stage I.-II., with gastric symptoms.
2 —
3 6 months.
4 N.
5 Y. No appetite, and pain in stomach after food.
6 N.
7 N.
8 No Th. 100t cc. T.A. 100'; 10045, 141'; marked pain in stomach.
9 Gastralgia, etc.
10
11
12 Treated with P.T.O. up to 1 cc.; wt. 100t. 100lb., gained 48lb.; then P.T. up to 1 cc.; wt. 100t. 100lb. "Feet splinted," and regained old energy, and lost gastric symptoms. I sent down to this patient living 400 miles from Sydney. Taught him how to give himself doses, no trouble.
Brother is control case. Died in 2 years of pulmonary tuberculosis. Since treated by me quite well.

101

- 11.07. 31. 100t. 25lb.
1 Stage I.-II.
2 —
3 Cough for 8 months; pleurisy 3 months ago; could feel "grating" in much pain.
4 N.
5 Y. Indigestion.
6 N.
7 N.
8 No Th. Baccol.
9 Pleurisy.
10 Influenza? in 11.07; sleeps badly.
11 4.
12 Treated with P.T.O. up to 1 cc.; gained 45lb.; then P.T. up to

1 cc.; 100t. 100lb., gained 45lb.; "feet splinted," eyes and sleep well; got painted on strength of 4.
Remained at work throughout treatment.

102

11. 30t. 48lb.
1 Stage I.
2 4.
3 4 months ago vomited blood on blood.
4 A.
5 B.
6 N.
7 N.
8 No Th. 100t cc. T.A. 100'; severe swelling of arm.
9-11
12 Treated with P.T.O. up to 1 cc.; in 1 week gained 45lb.; then P.T. up to 5 cc.
Feels quite well and strong; no cough.

103

- 11.07. 36. 100t. 25lb.
No family history.
1 Stage I.-II. or II.; marked splinted, etc.
2 —
3 Cough for 6 months; 1 year ago operated on for tuberculous disease of the chest, large vein left.
4 N.
5 Y.
6
7 1/2. 100' to 100'.
8 Th. G. 2 1/2 spum 100t. 141'.
9 Haemorrhage; several 1/2.
10-11
12 Treated with P.T.O. 100t cc. P.T.O. 100'; gained 45lb. in 4 months; in two months temperature gradually fell to normal; then P.T. sensitive; several reactions 100' to 100' up to 7 cc. (11.07). Dr. Griffiths continued treatment for 2 months, and then gave a rest because reactions continued. No cough and no phlegm. Had a rest at Manly on sea-coast for first month, but afterwards went to daily to work in his manufactory. He could not afford to do otherwise.

From January to February, 1908, several reactions (100°, 101°, 101°, 100° 3').

6.1.08. 1 cc. P.T. 102°

Three doses of same strength, 102° 4', 101°, 102° 4'.

Therefore treatment suspended for 3 months.

Then—

1.4.08. P.T.O. up to 1 cc. in 14 days; no reaction; then P.T. up to 255 cc. without any reaction, showing increased tolerance after a pause.

P.T. continued—

15.vi.08. 13 cc. P.T. 100° 110 glls.

21.vi.08. 39 cc. P.T. 100°

17.vii.08. 45 cc. P.T. 100°

2.viii.08. 33 cc. P.T. 100°

12.viii.08. 5 cc. P.T. 102° 4', 110 glls.

27.viii.08. 6 cc. P.T. 100° 4'

2.ix.08. 6 cc. P.T. 100° 4'

20.x.08. 7 cc. P.T. 99° 6', 110 glls.

25.x.08. 8 cc. P.T. 99° 6'

5.xi.08. 95 cc. P.T. 100°

11.xi.08. 110 glls.

Thus by increasing the doses, greater tolerance is induced.

The ultimate result was highly satisfactory. Hardly any phlegm and only slight cough in evening.

March, 1909. Looks and feels very well.

Sent two other cases for treatment.

104

12.07. 25. 60. 101b.

1 brother wt. 25 lb. ill 5 months. Father and father's brother 40.

1 R. 1-11. Diagnosed by other doctors.

2 —

3 No pain, symptoms.

4 X.

5 Y.

6 110-120

7 100°

8 No T.

9-11

12 Treated with P.T.O. up to 8 cc. in six weeks, wt. 72. 40b; then P.T. up to 75 cc. Dr. Griffiths continues treatment. Temperature gradually fell to normal and re-

mained so; pulse also improved greatly.

March, 1908. 875 cc. P.T. 98° 4'

In August, 1908, wt. 74. 87b.

104a

18. 90. 91b.

1 R. 1-11

2

3

4

5

6

7

8

9-11

12 Treated with Old T. up to 4 cc. gained 4 lbs. Feels better, looks better, and has hardly any cough.

105

21.07. 25. 100. 11b

Got a first class 11b the day before he came on and Dr. H. said "nothing like mother."

1 R. 1-11.

2 —

3 Cough for 6 months.

4 X.

5 R.

6 N.

7 N.

8 No Tx. Reacted to 100 cc. Old T. 100° 2'; but patient was hysterical; gave 105 cc. temp. 102° 4'; no more eruptions; swelling, headache, and herpes instant.

9-11

12 P.T.O. up to 95 cc; gained 4 lbs. in a month; and then P.T. up to 105 cc; wt. 54. 84b.

Treatment continued in 1908.

P.T. no reaction 11b.

10.1.08. 105 cc. P.T. 102° 4'; wt. 100. 64b.

10.1.08. 105 cc. P.T. 99° 4'.

Had intermittent attack of vomiting and severe diarrhoea; lost wt. 100. 14b.

P.T. was subsequently continued up to 9 cc.

2.viii.08. 9 cc. P.T. 98° 4'; wt. 100. 104b.

106

19. 1906.

B. (hospital case).

1 Stage I-II.

2 —→

3 ?

4 X.

5 Y. See 10.

6 224.

7 E.

8 Mo. Tl. 1001 Old T. 48'4"; 1002

Old T. 101'4"; yet gained 5 lbs.

9

10 Acute lead poisoning.

11

12 Treated with P.T.O. up to 725 cc.;

wt. 84; 8 lbs.

Pt. looks very well.

107

21.

18.07.

B. (hospital case).

1 Sent in as case of *myeloid tumour*.
Frontal headache, vomiting; no
defec. no dia; sight good; pupil
reacts (X) to optic reaction;
muscles power good; sensation
impaired; reflexes normal;
temp. normal; pulse 72; no signs
in lungs.

Treated 10025 Old T. 99'8"; local
reaction in arm; 10025 Old T.
101'; gained 5 lbs. in 2 months.

Improved greatly under tuberculin.

108 Still under treatment by Dr.
109 Griffiths, but though all are
110 improving, they have not yet
had treatment enough to do
much good.

Case 106. Miss Ma—g. 20. 1901.
Treated with P.T.O. up to 5 cc.,
no reaction; wt. lost 6 lbs.

Then P.T. up to 7 cc.; first 11 lbs.
Three weeks later, lost 15 lbs.;
gained 12 lb.

111

112

113

114

115 Rejected, and 113 and 114 sent to

116 have treatment.

117—See notes.

118—Error in diagnosis.

Case 113 is one of the most puzzling
cases I have ever seen. She came from
Tunnet. These two doctors diagnosed
tuberculous disease of knee joint; and
person then came to Sydney and saw a
surgeon, who confirmed diagnosis. I
then saw patient and was of the same
opinion, and prescribed at once to treat
her, thinking that the nature of the
case was certain. However, after a
few doses of P.T.O., which, though
rapidly injected, produced no effect at
all, I began to be sceptical as to the
existence of tuberculosis. I therefore
gave some doses of Old T., up to
1015 cc., with no effect in arm as in
joint or in system. I then applied
Calmette's tuberculin reaction, which
was also absolutely negative. Patient
is still living tuberculous, though I fear
the possibility of myeloid tumour of
the lower end of the femur. It may be
a chronic inflammation of knee-joint,
not tuberculous. There was no history
of injury, and no pain at first.

Two months later Dr. M. ascertained
leg for tumours of lower end of femur.

In case 116 I made a mistake in the
diagnosis, because I did not see enough
tuberculin, and perhaps also the patient
did not know how to read the thermo-
meter. At the first visit I diagnosed
pulmonary tuberculosis in Stage I., and
tuberculosis also on end of vocal cords.
But in testing with tuberculin the patient
said that temperature throughout was
normal. I gave as third dose 1005 cc.
Old T. There being no reaction, I
made an appointment for patient to
come, so that I might remove a portion
of ulcerating mass in larynx for micro-
scopical examination. Patient did not
come again, but went to another
specialist, who removed a portion of
mass, and in this mass giant cells
were found. The subsequent history
of the case I do not know. I did not
see the patient again, but I heard that
she was having tuberculin treatment by
Wright's method.

Eight are still under treatment.

Eighteen cases wanted to see dose,
but had so or less than a month's
treatment.

I have herein presented 115 cases in the first stage of pulmonary tuberculosis (including in Stage I. all cases of Stage I.-II.), so that this classification agrees with the system recently adopted at the Vienna Congress of 1907, and includes rather more advanced cases than those of Stage I. in the scheme of the Imperial Board of Health of Berlin. From 1902 to 1908 20 cases in the first stage which reacted to tuberculin have not been treated, and three cases had only two months' treatment. Two cases reacting to tuberculin in 1902 and 1903, respectively, would not have treatment. One is dead, dying a year later, the other was dying in 1906. These are "control" cases.

Of the 110 cases treated fully from 1902 to 1908, I know of the death of three cases: 1 of acute malaria, 1 of splenic anemia, and 1 the day after laparotomy for symptoms suggesting intestinal obstruction or acute pancreatitis. This last case died in New Zealand. I have done my best to keep in touch with all my cases, and I have not yet heard of a single case dying of pulmonary tuberculosis. In short, I know of two deaths among 25 early cases that refused tuberculin treatment, and I know of no deaths among the 110 that have had treatment. I know also that 90 out of these 110 are well and strong, and I would lay a wager about the others, giving odds. *At the worst I can claim success for tuberculin treatment in 80 per cent. of cases treated from 1902 to 1908.*

Another case, No. 52, Stage II., went to sanatorium in first stage, and in less than three months after being discharged as "cured" and "one of their best cases," he came to me when the disease was in the late second stage. This patient was in the first stage when he went to the sanatorium. The examining doctor told me so (see No. 52, Stage II.). I was able to help this poor fellow with tuberculin. Another case in the first stage refused to have tuberculin treatment and went to the sanatorium. There he developed acute pleurisy and nearly died. He had to leave the sanatorium, and then again came to me. He was nearly in the third stage, and I declined to attempt treatment with tuberculin. These are further "control" cases treated at the sanatorium which should damage the credit of sanatorium methods. The

truth is that sanatorium authorities are now trusting to tuberculin to restore confidence in sanatoria. This may be worldly wisdom, but it seriously confuses the issue upon which practical schemes for dealing with tuberculosis in the community essentially depend.

STAGE II

1901

- 1
 1. Stage II. (R. II., L. I-II., C. pleurisy).
 2 —→
 3 2 years.
 4 X.
 5 Y.
 6 50—80.
 7 f. 99.4—99.6.
 8 Th. G. 3; system 3.
 9 Pleurisy, extensive.
 10—11 A.
 12 Treated with T.R. up to 2 cc.; several reactions (100° F. over 100° F.).
 13 100 cc. 2 cc. T.R. 100° F.; several times pain in stomach and over liver, with vomiting, six hours after doses; cough much less, with phlegm; "was free from cough and phlegm for a fortnight."
 14 100. Wt. 90 lb.; great retraction of right side of chest; then P.T.O. to 1 cc. and P.T. up to 175 cc.; had intercurrent attack of pleurisy

and wt. fell to 80 lb. again (1908) under treatment.
 Has so far survived 7 years.
 1900—comparatively well.

2

1. R. I-II.; L. II.; severe pleurisy also over cardiac area.
 2 —→
 3 2 years.
 4 X.
 5 Y.
 6 110—120.
 7 f. and P. 100°—101°.
 8 Th. G. 5.
 9 Pleurisy on left side.
 10 Severe cardiac weakness and tachycardia.
 11 C.
 12 Treated with T.R. up to 17 cc.; when he left me I suspected that he had also tuberculous pericarditis.
 Died 1900; survived 6 years.

1902

- 3
 1. R. II., L. I-II.
 2 —→
 3 2 years.
 4 X.
 5 Y (poor effect).
 6 50—90.
 7 f. (99—100°).
 8 —
 9 —
 10 Faintings and anæmia.
 11 B—C.

- 12 Few doses of T.R.; then T.R.; lost cough and recovered strength and energy; sensitive, therefore pause; 103 T.R. up to 3.4 cc. in 1900; five years later was still well; gained 17 lb.; 80 lb.
 1907. Well.

4

1. R. II-III.; L. I-II.
 2 —
 3 5 years; "gave up all hope."

- 4 X.
5 Y.
6 80—92.
7
8 Th. G. 1.
9 Typical plastic bronchitis and haemorrhages.
10 Severe hysterical symptoms: fainting and tremors after a dose.
11 8.
12 Treated with T.R. up to 2.1 cc.; Sat. 511c.
*05. Wt. 92. alba.; cough and other symptoms practically gone; feels grand.
7.12.05. Wt. 92. alba.; no cough; one small haem.; appetite very good.
In 1904 T.E. up to 2 cc.; reactions (101°, 102°).
1905. Feels quite well; no symptoms; 100c.
1906. Quite well.
1911. Quite well.

5

- 9.02. 30. 70c. 511c.
1 R. L. H.; also pt. L. L. H.; effusion at right base.
2 —→
3 6 months at least.
4 X; very weak.
5 Y.
6 95—100.
7 99°.
8 Th. later.
9 Pleurisy.
10
11 1.
12 Treated from May to Sept., 1902, with T.R., T.O., and T.E.
2.6.02. 125 cc. T.R. 101° 5'.
2.6.02. 107 T.O. 101°.

- 26.01.02. 107 T.O. = 2 T.R. 101° 5'.
28.01.02. 107 T.O. + 3 T.R. 103° 4'. Sat. 511c.
31.01. 3.01. and 13.01. severe reactions (103° 2', 103° 4', 103°) with 4 cc. T.R. = 2 cc. T.O.; 2 cc. T.E. and 105 cc. T.E.
24.01. 1.01. T.E. 103° 4'.
1.02. 4 cc. T.R. 103° 4'.
8.12.02. 125 T.R. 101° 5'; pause for one month.
20.2.02. Sat. 511c.
20.3.02. 1 cc. T.E. 103° 4'; 105 cc. T.E. 100° 4' up to 5 cc. T.R.; then T.R. up to 1.5 cc.
9.05. Wt. 81. 411c.
13.05.02. Sat. 511c.
In 1904 sat.; gained 45 lbs.
Returned in February, 1906; temp. 100° 4'—102°, and mixed infection with Th. P.T.O. up to 1 cc.; then P.T. to 125 cc.; 70c. 611c.
24.01.06. Sat. 511c.
28.01.06. Sat. 611c.; under P.T.; then again sensitive.
3.3.06. 4 cc. P.T. 103°.
19.3.06. 125 cc. P.T. 103°; pause 48 days.
1.07. 155 cc. P.T. 102°; pause for 2 months.
13.10.07. P.T.O. up to 1 cc.; normal temperature; then P.T. with reactions up to 125 cc. under 125; lost 17 wt.
This patient remained well so long that she did not come to me again till the disease had advanced and a cavity had formed at right apex, and an obstinate mixed infection supervened. Her husband recognized too late his error in not sending her to be treated. She lived some distance from Sydney.
1909. Died in June, 1908.

1903

6

- 11.03. 25. 92c. 511c.
Sent by Dr. M.
1 R. L. H.; L. L. H.; pleurisy with effusion.
2 —→
3
4 X.
5 Y.
6 N.

- N.
No Th. 101 cc. T.A. 100° 5'.
21 Pleurisy.
12 Treated with T.R. up to 6 cc.; then T.E. up to 3 cc.; wt. 92c. 511c.
2.03. No cough, no phlegm, "breathing good now," "light feeling gone," able now to walk 20 miles, have done it several times; before could not walk at

All without distress, now working hard in office and feeling well.
1905. Quite well but should be tested.
1909. Quite well.

7

1903. 1 K. H.; L. L.-H.
2 →
3 18 months.

32.

4 A.
5 B.
6 N.
7 N.
8 Th. G. 2-2.
9-11 A.
12 Treated with T.E. for few weeks only; results: anxious; improved so much that he thought he did not need any more treatment.
Have not seen or heard of him since.

1904

8

1904. 26. Oct. 1903.

No family history.

1 R. H.; L. L.

2 —→

3 1 year.

4 N.

5 V.

6 Excitable (60 to 80) to 100.

7 / and P. 101", 100", 102", 103 1/2"

8 Th. G. 2-2.

9 Hemorrhage every morning.

10 35-50.

11 Hemorrhage.

12 A.

13 Treated with T.E. in 14 days hemorrhage stopped but returned through much less.

14.10.04. 8 cc. T.E. 100 1/2".

15.10.04. 9 cc. T.E. 101 1/2".

16.10.04. 1 cc. T.E. 100 1/2".

17.10.04. 1 cc. T.E. 100 1/2".

18.10.04. 1 cc. T.E. 100 1/2".

19.10.04. 1 1/2 cc. T.E. 100 1/2";

same for 3 weeks.

20.10.04. 1 cc. T.E. 101".

21.10.04. 1 1/2 cc. T.E. 100 1/2".

22.10.04. 1 1/2 cc. T.E. 100 1/2".

23.10.04. 1 1/2 cc. T.E. 100 1/2".

24.10.04. 2 1/2 cc. T.E. 101 1/2".

25.10.04. 2 1/2 cc. T.E. 101 1/2".

26.10.04. 2 1/2 cc. T.E. 101".

27.10.04. 3 cc. T.E. 101 1/2".

28.10.04. 4 cc. T.E. 102"; hemorrhage ceased; went up country, and was very well for about a year.

29.10.04. Night hemorrhage after severe exertion; then P.T.O. up to 9 cc. and P.T. up to 9 cc.

1908. Has been well ever since;

in this case, although hemorrhages occurred, even large doses with reactions did not cause him protracted hemorrhages.

Four years later. Well.

March, 1909. Still well.

9

1904. 26. Oct. 1903.

Sister, mother, and brother, sp. (see Nov. 17, Stage I.)

1 R. L.; L. H.

2 —→

3 6 months or more.

4 N.

5 V.

6 N.

7 N.

8 Th. later 100 cc. T.A. 100 1/2".

9

10 A.

11 Treated with T.E. up to 25 cc.; usually passed w.

12.04. Treated 100, 100 1/2"; treated further with P.T.O.; and then

P.T. up to 25 cc.; wt. 100. 820-24-100. P.T. 500; 100. 700.

9 months later:

4.07. P.T.O. 50-200; then P.T. up to 25 cc.; 11" fresh ever so much better after the dose.

Developed a septic inflammation of both ear and gum. A severe mixed infection followed. This patient

died during my absence in England. She had attacks of abdominal and pelvic pain (as I have been

told), and a nurse who knew her well said that there was something more than tuberculosis.

1909. Died in June, 1908.

10

10.04. 18. 8th. 5lbs.

No family history.

1 R. L.-H. : L. H. : extensive pleurisy at base.

2 —→

3 2 years spinalgia.

4 X.

5 V.

6 90.

7 F. 100'—101'.

8 No Th. Resisted.

9 Pleurisy.

10 First diagnosed as impacted renal calculus and sent to hosp. for operation.

11 L.

12 Treated with T.E. up to 12 cc. : gained 12lbs. ; wt. 9th. 14lbs. : then on to 2 cc. T.E.

December, 1907. 33 years aet., quite well and got well ; 1 advised testing, but patient was satisfied ; married in 1907.

February, 1909. Seen patient ; looking well.

Met her in street, carrying her baby. She looked well.

11

11.04. 25. 7th. 14lbs.

Named husband for 12 years who died 3 months ago.

1 R. H. : L. ?

2 —→

3 1 month.

4 X.

5 H.

6

7 F. and P. 100'—101', 102'—103'.

8 No Th. Resisted.

9 11.

12 Sent by her mother to Bukarest ; cough worse ; sent away again ; has refused to go and came to me ; treated with T.E.

28.vi.04. 51 cc. T.E. 104'.

27.vi.04. 53 cc. T.E. 99'.

2.vii.04. 65 cc. T.E. 104' ; then T.E. up to 85 cc. ; reactions (104', 101', 100', and 101') ; in September, although no dose was given, temp. 105' with rigor, headache, vomiting, diarrhoea, abdominal pain, and emaciation, we also had face puffy for 9 days ; lost 5lbs. but soon

regained in spite of reactions ; wt. 5th. 5lbs.

18.05. VI. 9th. 14lbs. ; gained 12lbs.

20.12.05. 2 cc. T.E. 100'.

24.12.05. 3 cc. T.E. 100'.

29.12.05. 4 cc. T.E. 101'.

5.i.06. 4 cc. T.E. 99'.

10.1.06. 5 cc. T.E. 99'.

15.1.06. 7 cc. T.E. 100'.

19.1.06. 9 cc. T.E. 100'.

24.1.06. 11 cc. T.E. 100'.

1.2.06. 13 cc. T.E. 100'.

While in hospital acquired a mixed infection ; signs of active trouble over upper R. lobe up to inter-scapular region ; temp. 104' in evening, normal to morning, for 10 days ; gives an intermittent impression of collapsed R. lobe of 1% volume ; and effect was diarrhoeic ; in 12 hours temp. fell to normal and did not rise again.

1908. Patient quite well.

1909. Very well.

12

12.04. 24. 8th. 14lbs.

No family history.

1 R. H. : L. 1.

2 —→

3 6 months ; influenza ?

4 X ; 8th. 14lbs.

5 13.

6 84.

7 F. (100'—101').

8 No Th.

9 Severe hæmorrhage, 1 year ; second 1½ years.

10

11

12 Treated with T.E. up to 1/2 cc. ; gained 10lbs. in 20 days ; then T.E. up to 1 1/2 cc. ; many reactions (100', 101'6', 102', yet gained 14lbs.

5.v.05. 45 cc. T.E.

4.v.05. 15 cc. T.E. 101'6'.

28.v.05. 19 cc. T.E. 104' ; passed.

15.11.05. Treated 100'—105'—101, 100'6' ; then treated with P.T. up to 4 cc.

8pt. 1908. Test 105, gained 12lbs.

7.vi.07. P.T. (X) up to 7 1/2 cc. ; wt. 106.

1908. Well. Has been working hard and travelling much since first course in 1904.

Well late in 1908.

13 ✓

- x.02. 14. 21. 24lbs.
Sent to hospital by doctor as typhoid fever.
1 R. L.; L. II.; with acute pleurisy.
2 —
3 2 months.
4 X; 21. 24lbs.
5 Y.
6 100, 130, 120.
7 / and F. (100"—103").
8 No Th.; reacted.
9 Hysteria.
10
11 —
12 Treated with P.T. up to '25 cc.
x.05. 21. 24lbs.; gained 22lb.
in one year, though growth at puberty accounts for part of this increase.
x.06. Reacted slightly to Old T., though she feels and looks quite well; in the hospital alone she gained 24lb., from 21. 24lb. to 35. 48lb.

14 ✓

- x.02. 21. 21. 18lb.
Also sent to hospital as typhoid fever by doctor.
1 R. L.—II.; L. II.; pleurisy.
2 —
3 III 3, 4, or 5 months; fever for weeks.
4 X.
5 Y.
6 90.
7 /, 100"—100 1/2" and F.
8 Th. G. 3; system 5/8; 100 cc. Old T. 100".
9 Haemorrhage; pleurisy.
10 Rheumatic pains; neurotic also.
11 A.
12 Treated for 4 or 5 months by doctor for rheumatism; then sent to hospital as "typhoid fever"; great loss of energy; treated with T.E. up to '25 cc.; then reactions, 100", test dose, 125", one reaction 125".
23.x.05. No phlegm and no cough, feels well; wt. 90. 48lb.; gained 24lb.; then P.T. up to '35 cc.; soon afterwards and was well; should be tested.

15 ✓

- x.04. 30. 80.
Sister and husband 40.
1 Stage II.?
2 —
3 Cough and fever, a year.
4 X; sucking child.
5 Y.
6 100—114.
7 N.
8 Th. G. 2 1/2.
9 Haemorrhage OJ.
10
11
12 Treated with T.E. to '05 cc.
x.02.02. '05 cc. T.E. to 100"; still wt. 80. 10lb.; then P.T.O. up to '6 cc.; wt. 80. 24lb.; could not leave home to have more treatment; not seen since; improvement very marked in every way.

16 ✓

- x.06. 35. 100.
No family history.
1 Stage I.
2 —
3 2 months.
4 X.
5 Y.; lost strength.
6 N.
7 N.
8 No Th. 100 cc. T.A. 100"; awful headache, loss of appetite, pains in back, cough worse, spasms 3/4.
9
10
11
12 Treated with T.E.; sensitive.
x.02.02. '05 cc. T.E. 100".
13.x.04. 2/25 cc. F.A. 98 1/4".
16.x.04. '05 cc. T.E. 100"; gave up treatment because his friends told him that reactions were dangerous; gained 24lb.

16a ✓

- x.04. 35. 21. 13 1/2lb.
Entered hospital, 20. 3lb.
1 Stage II.
2 —
3 11 weeks.
4 A.
5 B.

- 6 So.
7 N.
8 Th.; also not on T.A. 99.2';
1001, 101'; after test down gained
2 lbs. in 26 days.

- 9
10
11
12 Had few doses in hospital and later
P.T.O. up to 105; wt. now (1907),
116. 10 lbs.; gave up treatment;
gained 20 lbs. at first entrance to
hospital 35 lbs.

17 ✓

1904. 30. 101. 11 lbs.

- 1 Stage II.
2 →
3
4 X.
5 V.
6 Sp.
7 N (99%).
8
9
10 Severe rheumatic pains in r. knee,
elbow, and shoulder, after doses.
11

- 12 Treated with T.E. for one month,
then went to country; gained
2 lbs.; not seen since.

18 ✓

General Case.

- 20
1 Stage II.
2 →
3 6 months
4 A.
5 B.
6 N.
7 N.
8 Th. 93.

- 9
10
11
12 Decided to have tuberculin and
had a month's treatment with
T.E.; gained 2 lbs.; then per-
suaded by friends to have
sanguinaria treatment instead.
Went to the sanatorium and was
exhibited within a year as a case
of arrested pulmonary tuberculosis.
Within 3 years he died of pul-
monary tuberculosis.

1905

19 ✓

19.05. 30. 101. 11 lbs.

- Mother, wt. 49. 48.
1 R. II. L. II.
2 →
3 Cough 2 years, came out from
England for "weak chest" 23
years ago.
4 A.
5 B.
6 N.
7 N.
8 No Th. 1001 co. T.A. 1000-;
1005, 101'; spoon, 5).

- 9
10
11
12 Sensitive to tuberculin. First 3
doses of P.T. 101'. Still more on
with P.T. and reactions ceased.
26.1.06. 1375 co. P.T.
Felt quite restored to health and did
not think he needed more treat-
ment; wt. 101. 12 lbs.
Has kept well.

20 ✓

19.05. 48. 101. 10 lbs.

- 1 Stage II.
2 →
3 More than a year.
4 A.
5 V.
6 N.
7 N.
8 Th. G. 3.

- 9
10
11
12 Treated with E.T. up to 9 co.
Lost a few pounds (lost 6 lbs.),
but there was great improvement,
and cough and phlegm ceased.
"Felt so well."
Believes has to be well.

21 ✓

19.05. 26. 81. 11 lbs.

- Father 60, 1 brother 40.
1 R. II. L. I. II.
2 →

3 1 year and 9 months.

4 X.

5 B.

6 72-84.

7 N.

8 Th. G. 2 1/2.

9 Pleurisy.

10 Atrial abscess.

11

12 Treated with P.T.O. to 2 cc.; then T.O.A. to 4 cc.; then P.T.O. to 2 cc.; then P.T.; very sensitive.

21.05. 1075 cc. P.T. 95'.

24. 1075 cc. P.T. 104'.

26. 1045 cc. P.T. 103 1/2'; then pause till 11.00; then P.T.O. and P.T. to 175 cc.; wt. 86; 114.

12.07. T.B. up to 2 cc.

12.07. Wt. 86; 114.

Feels better, looks better, and has lost cough.

22

11.05. 12. 86.

No family history.

1 R. H. > L. H.

2 —

3 10 years' history.

4 X. Deadly weakness.

5 V. No appetite.

6 101.

7 101'—102'; gen. 99 1/2, 100'.

8 Th. G. 4.

9

10

11

12 Treated with P.T.O. to 1 1/2 gr.; many reactions at first and very sensitive; 4 months for course of P.T.O. to 2 cc.

1908. Extremely well.

(Died in July, 1908, of acute pneumonia within 3 days of onset.)

23

11.05. 11. 71. 94.

1 Stage II.

2 1/2

3 2 years.

4 X.

5 V. Ruined digestion with creasote, which he took for 3 years.

6 N.

7 N.

8 No Th. 100 cc. Old T. 100—; 105, 107'.

9

10 Alluminaria (Tub. H.)

11

12 Had open-air treatment and creasote and infusion. Doctor said "stick to creasote." Treated with T.B. up to 2 1/4 cc. No cough or phlegm. Used to get up two or three times in the night. Since treatment, has not to get up at all.

1908. Still well.

24

11.05. 76. 71. 84.

Father et. 43. 86.

1 R. H. > L. H.

2 —

3 1 year.

4 X.

5 V. Indigestion.

6 N.

7 N.

8 Th. G. 3 1/2.

9

10

11

12 Treated with P.T. up to 1 1/2 cc. gained 14 lbs. in a month.

3.11. 7 cc. P.T. 100'; then

treated with P.T. up to 75 cc.

The doctor who sent her to me consented to the result of this case.

1908. Well.

25

11.05. 22. 71. 116.

Mother suffers with chest.

1 R. H. 1. L. H. II

2 —

3 2 years. Night sweats for 4 years.

4 X.

5 B.

6 N.

7 1/2

8 Th. G. 2 3/4 116

9

10

11 1/2

12 Treated with P.T. to 125 cc.; gained 11 lbs.; wt. 84. Went away for a month.

25.11.06. P.T.O. to 1 cc.; then

P.T. up to 1 cc.; great improvement, lost cough and phlegm.

Went up country and lost sight of

25 ✓

VII.05. 25. 8th. 6th.

1 Stage II. (H. II. L. I-II.)

2 —→

3 Two years' cough. Three years ago treated by Dr. A. for pleurisy.

4 A.

5 V.

6 100.

7 N.

8 Th. G. G. Clamps. 3/8.

9-11

12 Treated with P.T. up to 100 cc.; then pause for 3 months.

XIII.06. P.T.O. to 8 cc.; T.O.A. to 5 cc.; then 3 v. 100. P.T. again up to 72 cc., and, finally, T.E. up to 35 cc.

Well, when last in 1927.

27 ✓

VII.05. 27. 9th. 11th.

Husband d.

1 Cystitis—probably Tals.

2 —→

3 Since birth of child 2 years ago.

4 A.

5 V.

6 86-96.

7 100 (100-101).

8 No Th. 100 cc. T.A. 100'4'; prolonged reaction for 24 hours.

9

10 Neuritic.

11 d.

12 Very sensitive to tuberculin; reactions 101-102'; then no reaction above 100'4'; then was persuaded to bend to general hygiene. Had only a few doses—should not be included.

History: known to be fairly well a year later.

28 ✓

— 28. 8th. 4th.

No family history.

1 4 years ago enlarged cervical glands; removed suppuration and large abscesses, wide over and above and below clavicles; discharged for 2 years.

2 —→

1 Wt. lost after abscess; now 8th. 4th.

2

3

4

5 No Th. Reacted severely to 100 cc. Old T.

29 ✓

VII.5. 29. 8th. 10th.

1 Enlarged glands of neck; glands of 1 side were removed extensively; now large and numerous glands on 1 side, as large as small hen's egg.

2 —→

3 Veno.

4-7. Nil; very fat.

8 No Th. 100 cc. T.A. 100'4'.

9-11

12 Treated with T.A. up to 7 cc.; at first with P.T., but very sensitive.

22. XII.05. 100 cc. P.T. 101'

29 is 101 cc. P.T. 100'6'.

15. X. 105 cc. P.T. 100'6'; then Old T. less reaction.

2. XI. 100 cc. Old T. 99'.

2. I.06. 100 cc. T. 99'. Pause then.

7. I.06. P.T.O. up to 100 cc.; T.O.A. up to 9 cc.; then C.T. up to 7 cc.

Had glands then removed by Dr. B.

Cervical Case.

4th. 11th. 4th.

1 Stage II.

2 —→

3 2 years.

4 A.

5 B. Dyspepsia.

6 N.

7 N.

8 Th. G. 5.

9 Hemorrhages.

10

11 d.

12 He felt well, and said he could not afford time to take treatment.

Meeting him again in street, urged him to be treated. He did not yield to my coaxing, and died within a year of 4.

1906

30

1906. 35. 12st. 2lbs.

- 1 Stage II. (R. II. & L. II.)
 2 →
 3 2 years.
 4 A.
 5 R.
 6 N.
 7 N.
 8 Th. G. 3. 5.
 9 Small hemorrhage.
 10
 11
 12 Previous treatment failed. Treated with P.T.O. to 10 cc.; wt. 12st. 2lbs.; then T.O.A. to 4 cc.; then P.T. up to 1 cc.
 13.07. Doubtful if started; slight reaction. T.R. up to 2.5 cc. from November 13 to December 18 without any reaction; wt. 12st. 9lbs.; no cough or phlegm or sputum.
 Feels and looks strong and well.
 April, 1909. Very well.
 Aug., 1911. Quite well.

31 ✓

1906. 44. 7st.

Elderly leather dyer; was weak with loss.

- 1 R. II. L. II.
 2 →
 3 18 months.
 4 N.
 5 R.
 6 N.
 7 N.
 8 Th. G. 3. 10.
 9 Pleurisy.
 10-11
 12 Treated with P.T.O. to 65 cc.; then T.O.A. to 35 cc.; and two doses of P.T. Improved; gained weight; but had trouble with her husband, and could not continue.

32

1906. 37. 10st. 4lbs.

No family history.

- 1 R. II. L. II. >
 2 →

- 3 Since Feb. 1904. Dr. Percy Kidd treated for throat in Sept., 1905.
 Sept. 1909. Fairly well.

- 4 A.
 5 R.
 6 N.
 7 N.
 8 Th. G. 3.4. 5.
 9 Hemorrhage and hemorrhage.
 10
 11 A.
 12 Treated for throat in 1905. Treated with P.T.O. up to 95 cc.; then T.O.A. to 35 cc.; then P.T. to 65 cc.; 10st. 4lbs. (normal wt. 9st. 6lbs.).
 vi.09. T.R. up to 2.5 cc.
 Two reactions with each dose of 25 cc. 10st. 4lbs.
 Feels well and looks well. July, 1908.

33

1906. 39st. 2lbs.

Fruit dealer.

- 1 R. II. L. I.
 2 →
 3 For years.
 4 N.
 5 Y.
 6 N.
 7 N.
 8 No Th. 100 cc. T.A.; 100 cc. 10st. 2lbs.
 9
 10 Rheumatic arthritis (knee).
 11
 12 Treated with P.T.O. to 4 cc.; then P.T. up to 25 cc.
 Much better; no cough. "Hardly feels anything in lungs now."

34

1906. 29. 7st.

- 1 R. II. > L. I.-II.
 2 →
 3 History in 1901; severe hemorrhage five weeks ago.
 4 N.
 5 R.
 6 60-65-100.
 7 N.
 8 No Th. Severe reaction.
 9 Hemorrhage. Op.
 10 Typhoid fever and appendicitis.
 11 A.

12. Sent by Dr. M. to Lithgow in 1901 or 1902.
Treated with P.T.O. up to 1 cc.; then T.O.A. up to 75 cc.; gained 10 lbs. in two months; then P.T. very sensitive.
13. 11.06. '05 cc. P.T. 104 W., 4 mo. after 1 = P.T.O. had no effect. Yet he says he "can walk more easily and breathe more freely since treatment." '04 cc. P.T. repeated 1 time, always 102'. Passed for 5 months; then P.T.O. to 1 cc. and P.T. without any reaction or trouble up to 4 cc.
Many reactions (102'4", 101', etc.). 1908. Was very well.
Feb. 1908. '0 cc. P.T. 118; wt. 50. 12 lbs. No cough.
Since. Writes that he is doing well.

35

- 11.06. 31. 30. 11 lbs.
1 K. H. L. H. = H.-H.
2 ———
3 Cough, 11 years.
4 A.
5 V.
6 10.
7 F. 94' = 100' = 100' = 100'.
8 T. G. 3. System 3.
9 Hemorrhages.
10 ———
11 ———
12. Treated with P.T.O. up to 9 cc.; then '05 cc. P.T. 100'; '02 cc. P.T. 100'.
11.07. Much less phlegm and no blood. New warts over R. lung; L. lung also strict; wt. 90. 12 lbs.
Not seen since.

36

- 11.06. 31. 30. 12 lbs.
No family history (N. 84. 1884).
1 K. H. (sister). L. H. (sister and brother).
2 ———
3 5 months. Influenza 9 months ago.
4 X.
5 Y. Bad.
6 90-100.
7 A. 100'.
8 T. G. 3-0.
9 Hemorrhages 3.

10. Insomnia.

11. 1.

12. Treated with P.T.O. up to 75 cc.; gained 10 lbs. Then on to 1 cc. P.T.O. and 75 cc. T.O.A.; then P.T. up to 6 cc.
During my absence in Tasmania, patient had a peculiar attack: severe headache and nausea with out vomiting. No stiffness at back of neck; had funny sensations; but her senses as soon as she lay head on pillow, and on opening eyes was giddy and staggered. For two days could not walk; would topple over; could not lift head from pillow. Patient is deaf, and there is loss of bone conduction. There is sometimes pain in the ears (Meniere's disease).
1908. Patient is greatly improved; no cough or phlegm or rales.

37

- 11.06. 31. 30. 11 lbs.
No family history (N. 101).
Worked in Telegraph office with a man who died of 4 and used to experiment all over the place.
1 R. H. L. L.-H.
2 ———
3 Hemorrhages 3 years ago; second, 10 weeks ago; third, 11 days ago.
4 X.
5 Y.
6 N.
7 N.
8 T. G. 3. System 3.
9 Hemorrhages 3-10-11.
10 Phlegm.
11 5.
12. Treated with P.T.O. up to 35 cc.; then T.O.A. up to 9 cc.; then P.T. up to 4 cc. Reactions:—
11.15.06. '05 cc. P.T.O. 102'.
11.16.06. '05 cc. P.T.O. 98'4".
11.17.06. '04 cc. P.T.O. 95'4".
11.18.06. '04 cc. P.T. 104'; within a week of 35 cc. P.T.O. and 9 cc. T.O.A.
1908. Head of him as well and back at work, having gained many pounds in weight and recovered all energy.
1909. Continues well.

38

v. ob. 28. 1st. 5th.
Brother and sister resided to T.

1 Dr. MacC. removed tub. glands of neck in August, 1905, now other glands enlarging on same side under sternum-mastoid and over scapula; also lungs R. I.-II.; cough, phlegm, short of breath.

2 —→

3 See above.

4

5 V.

6 M.

7 N.

8 No Th. 100 cc. T.A. 100'.

9 Pulm. T. and T. glands.

10

11 A.

12 Treated by Drs. M. L. D., who found swelling in lung. Treated with P.T.O. up to 1 cc.; then T.O.A. up to 7 cc.; then P.T. up to 15 cc.; moderate reactions. Went to Tasmania, did his two weeks in spite of advice, walked up Mt. Wellington; glands began to enlarge again.

11.07. T.H. up to 85 cc.; wt. 81. 47th.

In v. ob. enlarged glands softened, and was removed by Dr. MacC.

1908. Seems well, but has had much worry.

39

v. ob. 23. 12th. 5th.
No family history.

1 R. II.

2 —→

3 Heavy 10-months' cough; temp. 99.5; rigor; 9 weeks in bed; cough ever since.

4 A.

5 B.

6 N.

7 N.

8 Th. G. 2. Sputum 24.

9 Hemorrhages.

10-11

12 Before had 6 months open air by Dr. S. I., yet now active second stage; treated with P.T.O. up to 5 cc. Greatly improved, cough much less and much less sputum; looks to be in splendid health.

Said he could not afford time for more treatment; none's the pity.

40

v. ob. 29. 10th. 5th.
G. G.

No history is past, but sister and brother resided to T., and were treated.

1 R. II.; L. I.

2 —→

3 6 months.

4 X.

5 V.

6 S.

7 T. 99'.

8 Th. G. 2.

9-10

11 A.

12 Treated with T.T.O. and P.T. up to 1475 cc.

25.10.05. 475 cc. P.T. 100'.

3.11.05. 475 cc. P.T. 99'.

10.11.05. 55 cc. P.T. 98'.

17.11.05. 25 cc. P.T. 99'.

22.1.06. 10 cc. P.T. 100'.

Tested in 11.07. 103 cc. 104 T. 99'.

11.07. Treated with T.R. up to 2 cc. 35.11.05. 15 cc. T.R. 100', and ten days after this dose, opened index, etc.

After treatment, glands under, 1.25.

Oct., 1908. 12th. 5th.

March, 1909. Very well, no cough; wt. 120. 5th.

41

v. ob. 24. 9th. 2th.
1 R. II.

2 —→

3 Hæm. O., Christmas, 1904.

4 A.

5 B.

6 N.

7 N.

8 Th. G. 2.

9 Hemorrhage.

10

11 A.

12 Previous treatment: 5 years in New England, also in Yau.

Treated with P.T.O. 15 cc. reactions, 100'—100'.

Then T.O.A. 10 cc.; then P.T. up to 100 cc.; a few reactions (1903).

12.10.07. Tested 1005 cc. T.A.—015.—1. 105. 100'.

Further course, P.T.O. 10 cc.; then P.T. 10 cc.; without reactions.

Very well; no cough or phlegm; at work throughout treatment.

42

11.06. 25. 3at. 611a.
No family history.

- 1 Stage II.
- 2 —→
- 3 2 years.
- 4 A.
- 5 B.
- 6 N. (75-80 or 72).
- 7 N.
- 8 Th. G. 2.
- 9 11
- 12 Previous treatment for 6 months by creosote and inhalations, no better. Then P.T.O. to 39 cc., T.O.A. to 25 cc., and P.T. up to 1/4 cc. 1 position, 1.
- 1.11.06. '25 cc. P.T. 100/4'
- 6.11.06. '1 cc. P.T. 101'
- 15.11.06. '3 cc. P.T. 99'
- 21.11.06. '4 cc. P.T. 100'
- 30.11.06. '4 cc. P.T.
- 1.12.07. Treated 01
1908. Apparently well; wt. 100 lb., gained 15 lb.

43

11.06. 25. 811.

- 1 R. II.; L. II.
- 2 —→
- 3 1 year ago.
- 4 A.
- 5 B.
- 6 N.
- 7 J. 69-90/4
- 8 Th. G. 2.
- 9 11
- 12 Treated by open air for 2 years; yet disease progressed; then P.T.O. up to 1 lb.; sometimes slight (69/6-80/6), then T.O.A. to 1 cc. (100/4'), then P.T.
- 11.06. Treated two graduated times for patient to report; she gave stronger dose first; temp. 101°.
- 5.1.07. '21 cc. P.T. 100/6'
- 20.11.07. '2 cc. P.T. 99'; 70 lb. rylla. Lost a pound in weight, but greatly improved in health and physical signs.
- Hardly any cough or phlegm.

44

11.06. 25. 891.
1 R. II.
2 —→

- 3 Dating from childhood, 15 years old; numerous extensive chronic discharging sores which have left huge scars in skin, arms, neck, and nose. Hemorrhage from lungs in 1900.

- 4 X.
- 5 B.
- 6
- 7 J. 100', exp. after meals.
- 8 No Th.
- 9 Hemorrhage.
- 10 Little or diarrhoea and dysentery.
- 11 Had to give up work 4 years ago.
- 12 Treated with P.T.O.; treated on 99/0-100' after each dose, but increased emaciation; in a month. '1 cc. P.T.O. 99'; then 1.
- 24.10.06. '45 cc. P.T.O. 100'.
- 25.10.06. '6 cc. P.T.O. 98/4'.
- 1.11.06. '5 cc. P.T.O. 99/4'
- 15.11.06. '11 cc. P.T.O. 98/4'; gained 20 lb.; wt. 101; 48 lb.; then P.T.
- 22.11.06. '12 cc. P.T. 100'.
- 26.11.06. '10 cc. P.T. 100'
- 30.11.06. '10 cc. P.T. 99/2'.
- 5.12.06. '25 cc. P.T. 100'; then 100 lb., went home to Amstahle, got a very severe attack of dysentery, lasting some weeks, lost much weight; came to see me, sent him to Farnham, where he gradually recovered health and weight.
- 1.07. Further course P.T.O. to 99 cc. and P.T. up to 37 1/2; lost 10 lb.; never so heavy before; bowels now regular; before severe diarrhoea alternating with constipation (Feb. 5); now got better of constipation and no diarrhoea for 6 months.
- Looks poorer of health.

45

(100) 25. 811. 1100.

- 1 R. II.; L. I-II.
- 2 —→
- 3 Previous, Jan. 1905.
- 4 A.
- 5 V.
- 6 N.
- 7 N.
- 8 Th. G. 2.
- 9 11
- 12 Treated with P.T.O. 50 (100);

then T.O.A. to 25 cc.; then
P.T. to 750 cc.; 3 reactions (144°)
4.10.06. 225 cc. P.T. 100°.
10.11.06. 25 cc. P.T. 101°.
12.11.06. 4 cc. P.T. 100°.
2.12.06. 45 cc. P.T. 101°.
Treated 21.07. 1905 cc. T.R. 100°
—75 cc. 0.6 T.; 75 cc. 0.6 T.
100 for 100 times
1908. Well cured.

46

11.06. 27. 84. 118a.
1 Stage II.
2 ———
3 Cough for 8 years; losing a lb. a
week till treatment with tuber-
culin, then put on job.
4 X.
5 V.
6 S.
7 S. 100°, 101°, 101°.
8 No Th.
9-10
11 A.
12 Treated with P.T.O. Sensitive.
12.11. 1002 cc. P.T.O. 100°;
1021 cc. P.T.O. 100°; 1001 cc.
P.T.O. 99°.
12.11. 1005 cc. P.T.O. 100°
10.11. 1009 cc. P.T.O. 100°.
3.11. 1006 cc. P.T.O. 99°; wt.
84 lbs. No further reaction
with P.T.O. up to 2 cc.; Sat.
118a. then P.T. up to 6 cc.
Lost all the rheumatic pains from
which she had suffered as much
before tuberculin treatment.

47

11.06. 31. 100. 118a.
1 R. I-II; L. II.
2 ———
3 Since Jan., 1905.
4 A.
5 R. Large cysts.
6 N.
7 N.
8 Th. G. 3/4. Phlegm few.
9 Haemorrhages severe.
10-11
12 P.T.O. up to 2 cc.; T.O.A. up to
5 cc.; then P.T. up to 8 cc.
10.11.06. 375 cc. P.T. 104°.
22.11. 75 cc. P.T. 100°.
2.12.06. 35 cc. P.T. 98°.

17.1.07. 5 cc. P.T. 99°.
22.1.07. 7 cc. P.T. 100°.
2.12.07. 5 cc. P.T. 99°.
31.07. Severe haemorrhage occurred;
gave T.R. up to 2 cc.; no re-
action.
31.07. No riles; used to be worse
and violent; gained 14 lbs.; wt.
118a. 118b.
1908. Was very well, but had
recurrence of haemorrhage.
1909. Quite well. No further
haemorrhages.
Osteitis twice 2/3.
March, 1909. Is quite well.

48

11.06. 53. 101. 118a.
No family history.
1 R. II; L. I-II.
2 ———
3 Cough 6 months; phlegm, 2 years
ago.
4 X.
5 V.
6 N.
7 S. 99°-99°.
8 Th. G. 2 1/2; 3 1/2.
9 Haemorrhage 6/4 months ago.
10
11 A.
12 P.T.O. up to 2 cc.; then P.T.;
very sensitive.
25.1.07. 38 cc. P.T. 100°.
1.11. 1005 cc. P.T. 100°; then
T.R.
5.10.07. 1 cc. T.R. 100°.
11.10.07. 1 cc. T.R. 99°.
6.11.07. 145 cc. T.R. 100°.
13.11.07. 45 T.R. 100°; loss of
wt.; wt. 94 lbs.; over sensitive;
extreme to all forms.
31.07. Sat. 118a.
Coloured red; pt. could not afford to
eat; poor.
1909. Very well indeed. 102. 118a.
11.06.

49

11.06. 18. 101. 118a.
Sister Pp in 1906; father had many
kiss.
1 R. II; L. II.
2 ———
3 4 1/2 years.
4 X.

was in an advanced second stage, Th. G. 5 and wasted, pale and weak. I gave him P.T.O. up to '45 cc., gained 7 lbs.; then T.O.A. up to '2 cc.; then P.T. up to '475 cc.; wt. 90. 10 lbs.

He had to take work again on a ship and at intervals he came to see me. There was an immense improvement; he had hardly any cough; his appetite was very good. There was no relapse.

One has but to look on this picture and on that, and then explain the facts in the light of hope and reason. Unfortunately he had lapsed into a late second stage before I treated him, and one cannot be sure of results in such cases.

53

vi.08. 25. 100.
Buboes at 55 d. 12 years ago, and

lost two children of 8, 4, 14 mos. and 5 years.

- 1 R. H. > L. H.
 - 2 —
 - 3 Hemorrhage 8 years ago; in 1895 and in 1905 (O.).
 - 4 X.
 - 5 B.
 - 6 N.
 - 7 L.
 - 8 Th. G. 4. > 3/4. apnoea.
 - 9 Hæm. and pleurisy.
 - 10-11
 - 12 (Had open air in New Zealand); P.T.O. up to 175 cc., gained 10 lbs.; 100. 10 lbs.; then T.O.A. up to '75 cc.; then P.T. up to 25 cc. During treatment had very acute attack of pleurisy with high fever (105°) in July (1906), 1906, passed off in 10 days; afterwards no fever except with exertion.
- Went back to N.Z. apparently well.

1907

54

viii.07. 24. 70. 110.
One heehee d. larynx.

- 1 Stage II.
 - 2 —
 - 3 2 year; pleurisy 8 years ago.
 - 4 X.
 - 5 Y; 100.
 - 6 80-90.
 - 7 L.
 - 8 Th. G. 4.
 - 9 Hemorrhage.
 - 10 Asthma as a child, always a cough.
 - 11 1.
 - 12 Very sensitive to tuberculin; fast P.T.O. 1005. 1015° three times, 1st period 1015; cough better, less phlegm; increased to 1005 cc. P.T.O.; then T.O.A. up to '55 cc.; wt. 70. 8 lbs.; reaction 1015° went on to '55 cc. T.O.A. 800° one after other. Marked improvement, can get right tone in voice now, better in every way.
- Still under treatment.
T.O.A. contd. to 1005; then T.E. 10 '45 cc.
22.iii.08. 90 cc. T.E. 101°. 70. 10 lbs.

- 30 90 cc. T.E. 101°.
 - 31.v.08. 90 cc. T.E. 100°.
 - 22.v.08. 90 cc. T.E. 101°.
 - 3.vi.08. 5 cc. T.E. 99°. (Reduced dose.)
 - 5.vi.08. 7.66. 99°.
 - 13.vi.08. 9 cc. 99°.
 - 20.vi. 9.11. 99°.
 - 29.vi. 1.4. 102°.
 - 14.vii. 1.4. 100°.
 - 24.vii.08. 1.8. 100°.
 - 5.viii.08. 1.9. 99°. 70. 10 lbs.
- Few days later: feels well; absolutely no cough, and no phlegm. Physical signs: no rftm.
12.viii.08. 70. 70. 10 lbs.

55

- viii.07. 21. 90. 110.
No family history.
- 1 R. H.; L. H. > extensive pleurisy.
 - 2 —
 - 3 Cough, 6 months.
 - 4 Lost six pounds.
 - 5 B.
 - 6 N.
 - 7 N.

- 8 Th. G. 2-6 50 >.
9 Pusula is also with large excursions.
1009. Quins healed.

10

11

- 12 Treated with P.T.O. to 8 cc.; 1908.
Sibs.; then P.T. up to 375 cc.;
now very little cough, except after
cure; phlegm lost every day;
gradually improved.

Still under treatment.

March, 1909. Pusula of health.
Wt. lost 25 lbs.

56

- 11/07. 38. 100. 500.
Habit-keeper; no family history.

- 1 R. L-II; L. II. >.
2 —>
3 Fleury in Oct., 1904; hemorrhage
in Apr., 1905. 37
4 N.
5 E.
6 S.
7 N.
8 Th. G. 2.
9 Extensive pleurisy.
10 Bronchitis.
11 A.

- 12 Treated with P.T.O. to 25 cc.;
110. 500.; then P.T. up to 1 cc.;
110. 600.; no serious reaction
throughout. "No cough all the
week, though everyone else has
a cold"; can now sleep all night
from 12 to 8; used to sleep three
or four hours and then long for day.
This man has been busy through-
out with his hotel.

1908. Very well. Gained 12 lbs.

57

- 11/07. 32. 50. 300.
Father 46.

- 1 R. II; L. I.
2 —>
3 Fleury in 1899; hemorrhage one
month ago.
4 N.
5 Y.
6 N.
7 N.
8 Th. G. 2.
9 Hemorrhage.

- 10 Pains in joints and back after doses;
night sweats.

11 —>

- 12 P.T.O. up to 5 cc.; then P.T. up
to 375 cc.; several reactions;
100, 200, 300, 400, 500, 600, 700, 800, 900, 1000.
Each better, but will need careful
watching for some time; hardly
any cough or phlegm.

11/08. 475 cc. P.T. 100. 500.
500.12/08. 475 cc. P.T. 100. 500.
600.21/08. 475 cc. P.T. 100. 500. 800.
700.Treatment suspended for a time be-
cause there were three severe re-
actions to same dose.

Will need same treatment.

58

- 11/07. 22. 100. 200.

- 1 Severe tuberculous lesion of wrist
with soft stained red fluctuating
mass, no doubt containing caseous
material, tender and painful on
movement.

2 —>

3 Many reactions.

4 N.; 50 and 100; lost weight and
looked better.

5 Y.

6 N.

7 N.

8

9

10

11

- 12 P.T.O. up to 50 cc.; then P.T.
100. 1. 00. P.T. caused two re-
actions; sent home to Talmien
to rest; good improvement; the
forming mass has been absorbed
and the joint is much improved.

Went home with instructions for
further treatment.

1911. Went quite well.

59

- 11/07. 30. 100. 700.

1 R. L-II.

2

3 Cough 8 or 9 weeks; took medicine;
"nearly killed me; vomited it."

4

5

6

- 7
8 No Tx.
9-11 Hemorrhages.
12 Went to hospital; weight fell to 100 lb.; P.T.O. to 3 cc.; the left elbow very much; then P.T. up to 1 cc.; gave up treatment on account of pain.

60 ✓

- 1000 14 114-150.
1 R. H.; L. L.
2 —
3 2-3 months.
4 A.
5 V.
6 104.
7 5-100°.
8 Tx. G. 5.
9 Laryngeal cancer?
10 Hemorrhages; several hemorrhages, 30.
11 —
12 Treated with P.T.O. to 10 cc.; 114-150; then P.T. to 475 cc.; great improvement, but sensitive to pain.

61 ✓

- 1000 25 100-125.
1 R. H.; still swollen.
2 —
3 Sent to work by physician as anemic; possibly peritoneal ascites.
4 A.; bloodless.
5 V.
6 100-105.
7 5-100°-100°.
8 No Tx.
9 Marked pain.
10 —
11 —
12 P.T. to 8 cc.; in 4 weeks gained 15 lb.; in 2 months, pain normal and patient gained; P.T.O. up to 30 cc.; then P.T. up to 75 cc.; 60-110; lost all symptoms, and looks splendid; feet as well as eyes in life.

62

- 1000 45
This case has a special interest, at any rate from my point of view. The patient had been treated for Hodgkin's disease, and possibly he had this disease, but under the peculiar circumstances I had no

opportunity of examining his blood. When I examined him, there were enlarged glands in the neck on the right side and also under the clavicle and in the axilla. But I found also the spot test of the heart displaced to the left. At once examining the right pleura I found fluid, and made a rough sketch of skull area in my notebook. Although fluid in the pleura may occur in lymphadenoma, I was disposed to make the diagnosis of tuberculous pneumonia. At any rate I proposed to give a test dose of tuberculin, to which he readily assented. There and then I gave him 100 cc. of O.M. T.; he recorded the following temperatures: 100° 8', 101°, 102°, 100°, and next day 99°, 98° 4', 98° 4'. He certainly had tuberculous and probably tuberculous pneumonia. Before seeing me he had consulted two other doctors (Dr. M., Dr. J., Dr. F., and Dr. B.). After seeing me, he was advised to try another doctor (Dr. F.), who denied existence of fluid, in spite of tuberc. displacement of heart and hilum. He then returned to Dr. M., who also stoutly denied that there was fluid. Thinking that the truth must be with the majority, he gave me up, and I did not see him again till the day before I left Sydney, when it chanced that Dr. Griffiths was in my consulting room. He then narrated the sequel. Within two months of his first visit to me, Dr. M. proposed to tap the right pleura; at that time the fluid had increased so as to be recognized by symptoms as well as signs. The patient awoke in the night, and went to another doctor (Dr. H.M.), who drew off a large quantity of fluid; he came back to me and told the whole story of his own accord. I have not had any news of him, but I imagine Dr. Griffiths has been treating him with tuberculin, as I wished to do six months before, if he had remained with me; I have little doubt that there would have been no cause for tapping. However, I have not heard from Dr. Griffiths what the

blood examination showed. There was, however, little room for disagreement about the diagnosis; such mistakes can be avoided without using tuberculin.

1909. Lymphadenoma excluded.

63

vi. 07. 31. 7th. 1910.

No family history.

1 R. H.

2 —

3 Treated by Dr. L. for 1 year.

4 X.

5 V.

6 N.

7 L. 100' by day.

8 No Tb.

9 —

10 —

11 —

12 Treated with F.T.O. up to 15 cc.; 15 cc. P.T.O. 100'; then P.T. up to 15 cc.; 7th. 1910. Good! Fall of temperature to normal in 3 weeks; thereafter temperature absolutely normal.

Patient should be treated again this year.

64

20. 8th. 1910.

No family history, no syphilis.

1 R. H.; L. L. H.

2 —

3 1 month's pharyngitis a year ago; attended by Dr. C.

4 X.

5 B.

6 N.

7 —

8 Tb. G. 1.

9 Hemorrhages. 55.

10 Anemic regurgitation (rheumatic).

11 —

12 Treated with F.T.O. 100' up to 1 cc.; then P.T. up to 275 cc.; improved steadily, gained weight, strength, and energy.

1908. Still under Dr. Goffin's.

P.T. up to 75 cc. (Feb. 1908); no reaction.

Oct. 1908. Wt. 100 lb.; never so heavy; red out, 60 lb.; gained 20 lb.; no cough; very energetic; no different from what he was.

Treated in 1909. Kept. Treated again.

65

40. 6th. 1910.

9. 07.

1 Cyano chrome staining into left pectora, 6th, withdrawn by aspiration.

2 —

3 Indolent case.

4 X; 6th. 7th.

5 Y.

6 48.

7 L.

8 No Tb.; 100 cc. T.A.; 100 cc. T.A. 100'.

9-11 —

12 Treated with E.T.O. up to 2 cc.; wt. 50 lb.; 7th. 1910. Then P.T. up to 55 cc.; 5th. 6th. 7th. 8th. 9th. 10th. 11th. 12th. 13th. 14th. 15th. 16th. 17th. 18th. 19th. 20th. 21st. 22nd. 23rd. 24th. 25th. 26th. 27th. 28th. 29th. 30th. 31st. 32nd. 33rd. 34th. 35th. 36th. 37th. 38th. 39th. 40th. 41st. 42nd. 43rd. 44th. 45th. 46th. 47th. 48th. 49th. 50th. 51st. 52nd. 53rd. 54th. 55th. 56th. 57th. 58th. 59th. 60th. 61st. 62nd. 63rd. 64th. 65th. 66th. 67th. 68th. 69th. 70th. 71st. 72nd. 73rd. 74th. 75th. 76th. 77th. 78th. 79th. 80th. 81st. 82nd. 83rd. 84th. 85th. 86th. 87th. 88th. 89th. 90th. 91st. 92nd. 93rd. 94th. 95th. 96th. 97th. 98th. 99th. 100th. 101st. 102nd. 103rd. 104th. 105th. 106th. 107th. 108th. 109th. 110th. 111th. 112th. 113th. 114th. 115th. 116th. 117th. 118th. 119th. 120th. 121st. 122nd. 123rd. 124th. 125th. 126th. 127th. 128th. 129th. 130th. 131st. 132nd. 133rd. 134th. 135th. 136th. 137th. 138th. 139th. 140th. 141st. 142nd. 143rd. 144th. 145th. 146th. 147th. 148th. 149th. 150th. 151st. 152nd. 153rd. 154th. 155th. 156th. 157th. 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587th. 588th. 589th. 590th. 591st. 592nd. 593rd. 594th. 595th. 596th. 597th. 598th. 599th. 600th. 601st. 602nd. 603rd. 604th. 605th. 606th. 607th. 608th. 609th. 610th. 611st. 612nd. 613rd. 614th. 615th. 616th. 617th. 618th. 619th. 620th. 621st. 622nd. 623rd. 624th. 625th. 626th. 627th. 628th. 629th. 630th. 631st. 632nd. 633rd. 634th. 635th. 636th. 637th. 638th. 639th. 640th. 641st. 642nd. 643rd. 644th. 645th. 646th. 647th. 648th. 649th. 650th. 651st. 652nd. 653rd. 654th. 655th. 656th. 657th. 658th. 659th. 660th. 661st. 662nd. 663rd. 664th. 665th. 666th. 667th. 668th. 669th. 670th. 671st. 672nd. 673rd. 674th. 675th. 676th. 677th. 678th. 679th. 680th. 681st. 682nd. 683rd. 684th. 685th. 686th. 687th. 688th. 689th. 690th. 691st. 692nd. 693rd. 694th. 695th. 696th. 697th. 698th. 699th. 700th. 701st. 702nd. 703rd. 704th. 705th. 706th. 707th. 708th. 709th. 710th. 711st. 712nd. 713rd. 714th. 715th. 716th. 717th. 718th. 719th. 720th. 721st. 722nd. 723rd. 724th. 725th. 726th. 727th. 728th. 729th. 730th. 731st. 732nd. 733rd. 734th. 735th. 736th. 737th. 738th. 739th. 740th. 741st. 742nd. 743rd. 744th. 745th. 746th. 747th. 748th. 749th. 750th. 751st. 752nd. 753rd. 754th. 755th. 756th. 757th. 758th. 759th. 760th. 761st. 762nd. 763rd. 764th. 765th. 766th. 767th. 768th. 769th. 770th. 771st. 772nd. 773rd. 774th. 775th. 776th. 777th. 778th. 779th. 780th. 781st. 782nd. 783rd. 784th. 785th. 786th. 787th. 788th. 789th. 790th. 791st. 792nd. 793rd. 794th. 795th. 796th. 797th. 798th. 799th. 800th. 801st. 802nd. 803rd. 804th. 805th. 806th. 807th. 808th. 809th. 810th. 811st. 812nd. 813rd. 814th. 815th. 816th. 817th. 818th. 819th. 820th. 821st. 822nd. 823rd. 824th. 825th. 826th. 827th. 828th. 829th. 830th. 831st. 832nd. 833rd. 834th. 835th. 836th. 837th. 838th. 839th. 840th. 841st. 842nd. 843rd. 844th. 845th. 846th. 847th. 848th. 849th. 850th. 851st. 852nd. 853rd. 854th. 855th. 856th. 857th. 858th. 859th. 860th. 861st. 862nd. 863rd. 864th. 865th. 866th. 867th. 868th. 869th. 870th. 871st. 872nd. 873rd. 874th. 875th. 876th. 877th. 878th. 879th. 880th. 881st. 882nd. 883rd. 884th. 885th. 886th. 887th. 888th. 889th. 890th. 891st. 892nd. 893rd. 894th. 895th. 896th. 897th. 898th. 899th. 900th. 901st. 902nd. 903rd. 904th. 905th. 906th. 907th. 908th. 909th. 910th. 911st. 912nd. 913rd. 914th. 915th. 916th. 917th. 918th. 919th. 920th. 921st. 922nd. 923rd. 924th. 925th. 926th. 927th. 928th. 929th. 930th. 931st. 932nd. 933rd. 934th. 935th. 936th. 937th. 938th. 939th. 940th. 941st. 942nd. 943rd. 944th. 945th. 946th. 947th. 948th. 949th. 950th. 951st. 952nd. 953rd. 954th. 955th. 956th. 957th. 958th. 959th. 960th. 961st. 962nd. 963rd. 964th. 965th. 966th. 967th. 968th. 969th. 970th. 971st. 972nd. 973rd. 974th. 975th. 976th. 977th. 978th. 979th. 980th. 981st. 982nd. 983rd. 984th. 985th. 986th. 987th. 988th. 989th. 990th. 991st. 992nd. 993rd. 994th. 995th. 996th. 997th. 998th. 999th. 1000th. 1001st. 1002nd. 1003rd. 1004th. 1005th. 1006th. 1007th. 1008th. 1009th. 1010th. 1011st. 1012nd. 1013rd. 1014th. 1015th. 1016th. 1017th. 1018th. 1019th. 1020th. 1021st. 1022nd. 1023rd. 1024th. 1025th. 1026th. 1027th. 1028th. 1029th. 1030th. 1031st. 1032nd. 1033rd. 1034th. 1035th. 1036th. 1037th. 1038th. 1039th. 1040th. 1041st. 1042nd. 1043rd. 1044th. 1045th. 1046th. 1047th. 1048th. 1049th. 1050th. 1051st. 1052nd. 1053rd. 1054th. 1055th. 1056th. 1057th. 1058th. 1059th. 1060th. 1061st. 1062nd. 1063rd. 1064th. 1065th. 1066th. 1067th. 1068th. 1069th. 1070th. 1071st. 1072nd. 1073rd. 1074th. 1075th. 1076th. 1077th. 1078th. 1079th. 1080th. 1081st. 1082nd. 1083rd. 1084th. 1085th. 1086th. 1087th. 1088th. 1089th. 1090th. 1091st. 1092nd. 1093rd. 1094th. 1095th. 1096th. 1097th. 1098th. 1099th. 1100th. 1101st. 1102nd. 1103rd. 1104th. 1105th. 1106th. 1107th. 1108th. 1109th. 1110th. 1111st. 1112nd. 1113rd. 1114th. 1115th. 1116th. 1117th. 1118th. 1119th. 1120th. 1121st. 1122nd. 1123rd. 1124th. 1125th. 1126th. 1127th. 1128th. 1129th. 1130th. 1131st. 1132nd. 1133rd. 1134th. 1135th. 1136th. 1137th. 1138th. 1139th. 1140th. 1141st. 1142nd. 1143rd. 1144th. 1145th. 1146th. 1147th. 1148th. 1149th. 1150th. 1151st. 1152nd. 1153rd. 1154th. 1155th. 1156th. 1157th. 1158th. 1159th. 1160th. 1161st. 1162nd. 1163rd. 1164th. 1165th. 1166th. 1167th. 1168th. 1169th. 1170th. 1171st. 1172nd. 1173rd. 1174th. 1175th. 1176th. 1177th. 1178th. 1179th. 1180th. 1181st. 1182nd. 1183rd. 1184th. 1185th. 1186th. 1187th. 1188th. 1189th. 1190th. 1191st. 1192nd. 1193rd. 1194th. 1195th. 1196th. 1197th. 1198th. 1199th. 1200th. 1201st. 1202nd. 1203rd. 1204th. 1205th. 1206th. 1207th. 1208th. 1209th. 1210th. 1211st. 1212nd. 1213rd. 1214th. 1215th. 1216th. 1217th. 1218th. 1219th. 1220th. 1221st. 1222nd. 1223rd. 1224th. 1225th. 1226th. 1227th. 1228th. 1229th. 1230th. 1231st. 1232nd. 1233rd. 1234th. 1235th. 1236th. 1237th. 1238th. 1239th. 1240th. 1241st. 1242nd. 1243rd. 1244th. 1245th. 1246th. 1247th. 1248th. 1249th. 1250th. 1251st. 1252nd. 1253rd. 1254th. 1255th. 1256th. 1257th. 1258th. 1259th. 1260th. 1261st. 1262nd. 1263rd. 1264th. 1265th. 1266th. 1267th. 1268th. 1269th. 1270th. 1271st. 1272nd. 1273rd. 1274th. 1275th. 1276th. 1277th. 1278th. 1279th. 1280th. 1281st. 1282nd. 1283rd. 1284th. 1285th. 1286th. 1287th. 1288th. 1289th. 1290th. 1291st. 1292nd. 1293rd. 1294th. 1295th. 1296th. 1297th. 1298th. 1299th. 1300th. 1301st. 1302nd. 1303rd. 1304th. 1305th. 1306th. 1307th. 1308th. 1309th. 1310th. 1311st. 1312nd. 1313rd. 1314th. 1315th. 1316th. 1317th. 1318th. 1319th. 1320th. 1321st. 1322nd. 1323rd. 1324th. 1325th. 1326th. 1327th. 1328th. 1329th. 1330th. 1331st. 1332nd. 1333rd. 1334th. 1335th. 1336th. 1337th. 1338th. 1339th. 1340th. 1341st. 1342nd. 1343rd. 1344th. 1345th. 1346th. 1347th. 1348th. 1349th. 1350th. 1351st. 1352nd. 1353rd. 1354th. 1355th. 1356th. 1357th. 1358th. 1359th. 1360th. 1361st. 1362nd. 1363rd. 1364th. 1365th. 1366th. 1367th. 1368th. 1369th. 1370th. 1371st. 1372nd. 1373rd. 1374th. 1375th. 1376th. 1377th. 1378th. 1379th. 1380th. 1381st. 1382nd. 1383rd. 1384th. 1385th. 1386th. 1387th. 1388th. 1389th. 1390th. 1391st. 1392nd. 1393rd. 1394th. 1395th. 1396th. 1397th. 1398th. 1399th. 1400th. 1401st. 1402nd. 1403rd. 1404th. 1405th. 1406th. 1407th. 1408th. 1409th. 1410th. 1411st. 1412nd. 1413rd. 1414th. 1415th. 1416th. 1417th. 1418th. 1419th. 1420th. 1421st. 1422nd. 1423rd. 1424th. 1425th. 1426th. 1427th. 1428th. 1429th. 1430th. 1431st. 1432nd. 1433rd. 1434th. 1435th. 1436th. 1437th. 1438th. 1439th. 1440th. 1441st. 1442nd. 1443rd. 1444th. 1445th. 1446th. 1447th. 1448th. 1449th. 1450th. 1451st. 1452nd. 1453rd. 1454th. 1455th. 1456th. 1457th. 1458th. 1459th. 1460th. 1461st. 1462nd. 1463rd. 1464th. 1465th. 1466th. 1467th. 1468th. 1469th. 1470th. 1471st. 1472nd. 1473rd. 1474th. 1475th. 1476th. 1477th. 1478th. 1479th. 1480th. 1481st. 1482nd. 1483rd. 1484th. 1485th. 1486th. 1487th. 1488th. 1489th. 1490th. 1491st. 1492nd. 1493rd. 1494th. 1495th. 1496th. 1497th. 1498th. 1499th. 1500th. 1501st. 1502nd. 1503rd. 1504th. 1505th. 1506th. 1507th. 1508th. 1509th. 1510th. 1511st. 1512nd. 1513rd. 1514th. 1515th. 1516th. 1517th. 1518th. 1519th. 1520th. 1521st. 1522nd. 1523rd. 1524th. 1525th. 1526th. 1527th. 1528th. 1529th. 1530th. 1531st. 1532nd. 1533rd. 1534th. 1535th. 1536th. 1537th. 1538th. 1539th. 1540th. 1541st. 1542nd. 1543rd. 1544th. 1545th. 1546th. 1547th. 1548th. 1549th. 1550th. 1551st. 1552nd. 1553rd. 1554th. 1555th. 1556th. 1557th. 1558th. 1559th. 1560th. 1561st. 1562nd. 1563rd. 1564th. 1565th. 1566th. 1567th. 1568th. 1569th. 1570th. 1571st. 1572nd. 1573rd. 1574th. 1575th. 1576th. 1577th. 1578th. 1579th. 1580th. 1581st. 1582nd. 1583rd. 1584th. 1585th. 1586th.

67

18.07. 28. 7th. 12th.

A case of mother cerebral.

1 R. I. L. H.: I lectured upon this case, making diagnosis provisional of latent disease, scabs and perhaps patent dactyl. Bone: very marked third year pulmonary artery and diaphragm. At 18 months I had system examined. Between 18th and 22nd there was well-marked pulmonary intercostal. Moreover, I was determined to show that tuberculosis was not contraindicated in cases of heart disease. The boy had also had pulmonary hemorrhage.

2 —

3 4 months.

4 N.

5 R.

6 N.

7 C.

8 Th. G. 4.

9

10 Congenital heart disease: treated with P.T.O. up to 100 cc. (100%) he had a hemorrhage followed by cough 100%, when on P.T.O. was given: in two months he gained 10 lb.; and in another two months another 10 lb. (wt. 94 lb.). Thus even in this bad form of heart disease, tuberculosis did good.

68

18.07. 41. 100. 5th.

Mother, et. 42, 5th, also under.

1 R. H., I.

2 —

3 Was sent to Australia by Sir A. Clarke twenty years ago; then broke down again, went to New Zealand; each year had an attack, got rid of cough and put on weight. This is the first case in Stage II. in which disease has been long at a standstill, though he has had no tuberculin treatment.

4 A.

5 R.

6 N.

7 N.

8 No Th. 100 (100).

9 11

12 Treated with P.T.O. up to 100 cc.; then P.T. up to 6 cc.

69

18.07. 22. Oct. 1914.

Hospital case.

1 Sent in at "gastritis or gastric ulcer." R. I., well-marked signs; also enlarged gland in neck in front of and under sternomastoid, L.

2 —

3 2 years.

4 N.

5 V. anorexia, frequent and severe vomiting after food, like symptoms of nervous dyspepsia, constipation. Dr. W. had diagnosed appendicitis and had operated, but no better.

6 Th.

7 C. to N.

8 No Th. 100 (Old T. 98%) 100; on Old T. 100%: swelling in area and cervical gland.

10 Diarrhoea: irregular menstruation: from 13 to 17, regular, from 17 to 18, amenorrhoea; since 18, every five months old tuberculosis was given; menarche in October 1909, and again in November — first time for first year.

11 2.

12 Treated with P.T.O. up to 75 cc.; gained 25 lb.; wt. 45 lb.; no vomiting, appetite much better; then P.T. up to 175 cc. remarkable improvement, looks now gay and robust; mother very pleased. March, 1910. Looking well, but excited; and now having father constant.

70

18.07. 24. Oct. 1914.

1 R. I. H., I. H.

2 —

3 Cough 8 or 10 months: ending in vomiting.

4 N.; lost 45 lb.

5 V.

6 100; intermittent.

7 C. 99%.

8 Th. G. 5; spum. 7.

9 Hemorrhage.

10

11

12 Not seen any doctor before; yet advanced disease; treated with P.T.O. up to 6 cc.; lost 45 lb.; probably would have lost more if he had not had tuberculosis; 1911

he feels much better, indeed quite well; cough much better and phlegm less.
Still under intensive treatment by Dr. Goldfarb.

71 ✓

44. 71st, 61st.

1 Stage II.

2 —→

3 Began on infusions Oct., 1907; phlegm, 11 years ago, recovered and passed first-class file for A.M.P. Society.

4 A.

5 B.

6 N.

7 N.

8 Th. G. 5.

9 11.

10 Had been with in country; put on weight and recovered appetite; is now under intensive treatment; doing well.

1909. Very good improvement.

72 ✓

44.

1 R. H. 5.

2 —→

3 Cough 3 months.

4 N.

5 Y.

6 So.

7 99.

8 Specimen 11.

9

10 Chr. (oxid) 11st.

11

Under treatment; has improved.

1909. Is very well. Recommended

to go to the last week (March).

73 ✓

44. 71st, 61st.

No family history.

1 R. H. 5th.

2 —→

3 Cough for some time.

4 N.

5 Y.

6 N.

7 N.

8 N.

9 N₁ Th. 100' 6"; T.A. 100' 1

100' 7"; 100' 100' 12"; cough worse.

11

12 Nausea and vomiting.

13

14

Under treatment.

P.T.O. up to 19 cc. Then P.T. up

to 775 cc.; several severe re-

actions 100', 100' 12', and 100' 12'

100' 12' = 775, 100' 12'.

Tubercles, 100'. Vented; did not

react to 100 cc. Old T.

74

44. 71st, 61st.

1 cc.

1 Stage I. Tuberculous peritonitis. Pain in abdomen, esp. right side; gastric pain; vomiting; diarrhoea, dirty tongue, constipation, white diarrhoea.

2 —→

3 2 years.

4 N. 1st. 6/10.

5 Y.

6 Y.

7 Y.

8 Y.

9 N₁ Th. 100' 15" OM T. 100' 12";

100', 100' 12'; 100', 100' 12'; there-

after normal for 3 days, morning

and evening.

11

12 Hygienic and satisfactory.

13

14 Treated with P.T.O. up to 15 cc.;

gave 5/10.

Tubercles in hospital. Went up

the country. No more since.

STAGES II-III.

1902

1 ✓

28. 54.
 1 R. I.-II.; L. II.-III. Extensive
 pleurisy with signs over cardiac
 area.
 2 ———
 3 2 years.
 4 X.
 5 V. Post apy. 1 month.
 6 110-120.
 7 C.
 8 Th. G. 3.
 9 Pleurisy.
 10 Cardiac weakness.
 11 A.
 12 Treated with T.E. up to 17 cc.

Greatly improved; but very weak and persistent tachycardia. I did not expect him to live another year, but he lived till middle of 1907. His brother (also Stage II, later) is still alive and having further treatment. He has had no treatment since 1900—eight years ago.

2 ✓

15. 76. 1114.
 Mother dp, only brother dp, only sister
 resided (1906).
 1 R. I.-II.; L. II.-III.
 2 ———
 3 10 months.
 4 X.
 5 R.

- 6 116-138.
 7 V. 100°-102°-103°.
 8 Th. G. 2. M. 4.
 9 Hemorrhage.
 10 ———
 11 A.
 12 Treated at first with ant., then T.E.
 up to 1 cc.; wt. 84. 1910. Great
 improvement, no cough or phlegm.
 Th. disappeared from system.
 17.03. Went to show, got a severe
 chill and relapse, and spinal in-
 fection (S.).
 21.07. Gave T.E. up to 1.5 cc.
 Many reactions, 100°, 101°, 99°
 pulse 90 and wt. 94. 1911.
 24.04. Gave T.E. up to 2.2 cc.;
 wt. 94. 1912.
 21.05. Wt. 94. rills. No cough,
 no phlegm.
 January, 1908. No cough, no spu-
 rum; fat and strong.
 April, 1909. Treated with Old T. up
 to .04 cc. No reaction.
 Again, in 1907, when question of his
 going to a public school had to be
 settled, I tested him again. Old
 T. up to .03 cc., no result.
 Dr. M., the family doctor, and an
 enteric script, wrote to me in 1904,
 and described the grave condition
 of his lungs—"litera scripta ma-
 net" is my pocket. In this case
 also we have the "controls" in the
 mother and brother.

1909. Quite well. *W. J. L.*

1903

3 ✓

14. 76. 1188.
 1 R. II.-III.; L. I.-II.
 2 ———
 3 Pleurisy, 8 years ago; consumption
 for 14 years.
 4 A.
 5 B.
 6 So. Good.
 7 N.
 8 Th. G. 3.6.
 9 Pleurisy.

- 10 ———
 11 A.
 12 Treated with T.E. up to 1.3 cc.;
 no reactions, but severe with T.E. 1
 cc. T.E. had no effect; yet 105°
 cc. T.E. raised temp. to 107°;
 pt. was much improved, but could
 not remain away from home any
 longer.
 Had no news of her date—good or
 bad. Certainly should have had
 more treatment.

4

- vi. 01. 31. 7th. 10th.
 1 R. H.; L. H.-III.
 2 —→
 3 2 years.
 4 X. 7th. 10th.
 5 V.
 6 25-34.
 7 N.
 8 Th. G. 3. > 25 spores.
 9 Haemorrhage.
 10
 11 2.
 12 Treated with T.E. up to 1 cc., then T.E. Haemorrhage began with "4 cc. (101', 102', 103', 104'). Pains for 3 weeks.
 13.04. T.E. up to 1 cc.
 14.04. 1 cc. T.E. var.
 22.04. 2.1 cc. T.E. (101' 2') var. little cough or phlegm—normality (101').
 2.5.04. 1.0 cc. T.E. (101' 2') wt. 84. 10th.
 8.5.04. 2.1 cc. T.E. (101' 2') wt. 84. 11th.
 31.01.04. 7th. 10th. 11th. gained (101').
 10.0. Was well. Worked in office during treatment and after 10th.
 10.0. Was well.

5

- vi. 01. 31. 8th. 11th.
 No family history.
 1 R. H.-III.; L. 1-II.
 2 —→
 3 2 years.
 4 X.
 5 V.
 6 34-40.
 7 N.
 8 Th. G. 3%. Spores 24.
 9 Infiltration of one vocal cord; loss voice.
 10
 11 1-2.
 12 Treated with T.E. up to 2 cc. in 1906 of severe reaction (101', 102', 103', 104', 105', 106', and great weakness; passed 10th.; not enough of expectoration, appetite good, and now plenty of energy; able to take severe work.
 1.01. Reacted to tuberculin; gave P.T.S. up to 1 cc., and
 12.01. T.E. up to 1.25 cc. (vi. 01. 11.01. Reacted; then P.T. up to 25 cc. (101'); wt. 90. 23th. Looks well and feels well. Need less to say, before he came to me, he had been treated by several doctors, but none of them by open-air methods, etc., did any real good.
 1900. Treated in Oct., 1908, did not react to 101 (11) T.
 1910. Had a small haemorrhage.

6

- vi. 01. 22. 10th. 13th.
 No family history; pains.
 1 R. H.-III.; L. II.
 2 —→
 3 1 year.
 4 X.
 5 V; pains.
 6 N.
 7 N.
 8 Th. G. 3 clumps M.S.; spores 4 capsid.
 9 Tuberculous laryngitis; pleurisy.
 10 Pains.
 11 2.
 12 Treated with T.E. up to 1.5 cc.; then T.E. up to 1.75 cc.; pain advised because 1.75 cc. T.E. caused 101' 2'; during treatment recovered voice, which he had lost

1904

completely for two months before he came to me. At first he gained 10th, but then lost 10th. In a few months very little cough ("not more than" 1), and system a decided improvement instead of half a capsid. Three months before he came to me, he saw Dr. S. J., who sent him up the country. Dr. S. J. said he was in an early stage, that there was "nothing to worry about." In two months he certainly gained 10th, but he lost his voice from tuberculous laryngitis, and the disease advanced from its early stage to a late second stage. I advised him to come to me again in three months, but instead he gave way to his old habit and went downhill. He was alive late in 1907.

7 ✓

46.04 20. 7th. 1914.

No family history.

1 Stage II.-III. = III. (06)

2 →

3 Said to be 5 months.

4 N.

5 V; poor diet.

6 100-120.

7 F. 102°-101°.

8 Th. G. 8. M.P. sputum 54.

9

10 Diarrhoea (T.) for six weeks and more.

11

12 At first treated with three intra-venous injections of collargol (15 + 5 cc.); then P.T. up to 35 cc.; in three months temperature normal. Improved enormously. Then sent her to maintain for a rest. Here she was informed by word—or at least by her mother—that I considered her case as hopeless; her brother came to see me and asked me if I had told the nurse so. I had told the nurse that she was in a late stage, so that she should adopt the proper precautions. I have not seen the patient or her parents since, professionally, though I passed the patient in the street late in 1907.

8 ✓

x.04 20. 9th. 1914.

1 Stage II.-III. = III.

2 →

3

4 N.

5 V.

6 90-100; even 120 without tubercula.

7 100 F. (100-102°).

8 Tuberculous laryngitis.

9

10

11

12 Treated with T.E. for few doses; then T.R. up to 3 cc.

26.x.04 725 cc. T.R. 105.4°; probably into vein.

4.x.04 2 cc. T.R. 98.4°.

10.x.04 3 cc. T.R. 101.4°.

18.x.04 3 cc. T.R. 103.8°; but later temperature rose to 104° for

six weeks; then P.T.O. up to 4 cc.; gave up treatment. Well in 1906.

9

10.04 21. 9th.

Mother, et. 37. 66.

1 R. II.; L. II.-III.

2 →

3 3 years ago haemorrhage.

4 A.

5 B; good appetite.

6 N.

7 N.

8 Th. G. 2.

9

10

11

12 Treated with T.E. up to 4 cc.; lat. 1914; lost wt.; very hot weather (105°) and had few erythema; tested six months after; tested 1005 (Th. G. 100°); then gave P.T. up to 5 cc.; wt. 30 1914.

2.x.06. Tested again, 1001, 102, 102, all negative; can play tennis now all the afternoon; no cough and no phlegm.

Came to see me late in 1907. Quite well.

1909. Well.

10 ✓

20. 7th. 1914.

No family history.

1 Stage II.-III.

2 →

3 For a year; 6 months night sweats.

4 N; 7th. 1914.

5 V.

6 N.

7 N.

8 Th. G. 3-5; phlegm 24 (100 cc.).

9 Home.

10

11

12 Treated with P.T. up to 15 cc.; some severe reactions, 105.5°, 104°, 102°, 105°, 103°, 102°; still went on gaining weight and improving till he weighed 100 lbs.; gained 18 lbs.; looked a different man after treatment in spite of reactions; and has been at work ever since.

Well at end of 1907.

11 ✓

39 7th 1876.

104.

No family history.

1 Stage II. With tuberculous testis.

2 →

3 Two years.

4 N.

5 V.

6 N.

7 N.

8

9 Very severe hemorrhage; trans-

fusion.

10-11 A.

12 Had been sent up country by Dr. F.; had a severe hemorrhage; testis still discharging; treatment with P.T. up to 14 cc.; abscess of testis healed completely; then T.E. up to 35 cc. (var. 8); sensitive but gained wt.; gained 8 lbs.; 5th. 3 lbs.; then P.T. up to 105; patient left off coming to his rooms that I can understand. His brother wrote to testify to the great improvement in his brother. Head aching since.

Control ✓

70 Oct. 1910.

104.

No family history.

1 R. II, III; L. II.

2 →

3 Cough 2 years.

4 N.

5 V.

6 N.

7 N.

8 Th. 2.

9 Many hemorrhages.

10

11

12 Saw Dr. W. and L. (a Hyatt), sent to Sydney; saw Dr. S. J., sent to country; saw Dr. H. at Orange; then Armistead, Glenbrook, Tenterfield; also Dr. G. W., Dr. R., and Dr. P.; put one of my patients and came to me; I decided to try tuberculin. However, he went away, and in a few weeks was advertising himself as a quack remedy, "Eucalen," which had "cured" him. Within 9 months he was dead.

1905

12 ✓

32 5th 1876.

109.

1 R. II, L. II-III.

2 →

3 12 years ago pleurisy and pneumonia; cough for five years; hem. in 1901.

4 N.

5 V.

6 100-120.

7 N.

8 Th. G. 2; much phlegm.

9 Asthma.

10 Appendicitis; severe pneumonia.

11

12 Treated with few doses of T.E. and T.R. (1975 cc.); then P.T. up to 7 cc. (Sat. 100a.); no asthma at all, greatly improved, appetite better, and sleep better. Looks far better.

4 →

5 1½ years.

6 N.

7 V.

8 80-100; 112 v. reaction.

9 N.

10 Th. G. 4.

11 Hemorrhages.

12

13

14 Before treatment went to Narrabri and gained 10 lbs.; yet now stage II-III; treated with P.T. A. up to 9 cc.; then P.T. up to 9 cc.; 100. Reacted; further treated with T.O.A. up to 14 cc.; then P.T.O. up to 9 cc.; then P.T. up to 1 cc. 1107. Tested, 1005, 1025, 1025, 1045, all negative.

Dec. 1907. Wt. 102. 6 lbs.; seems and feels quite well. Gained 2 lbs.

13 ✓

47 5th 1876.

105.

Mother, *et.* 35-40.

1 R. II; L. II-III.

1105.

1 R. II-III; L. I-II.

2 →

3 3 years.

14

54 5th 1876.

- 4 X.
5 V.
6 N.
7 N.
8 Th. G. 3. Sputum 30.
9 Hemorrhages.

10
11 A.

- 12 Treated with P.T. up to 375 cc.
Many reactions, no cough for 2 nights, no sputum; wt. 82. 7 lbs.; pulse on account of severe reactions.
1906. Moderate relapse; treated with P.T.O. to 1 cc., T.O.A. to 25 cc., and P.T. to 7 cc.; wt. 82. 7 lbs.; sputum more about a teaspoonful in 24 hours; no hemorrhages; can walk up hill; before treatment could hardly walk any distance; appetite very good. Heard of patient late in 1907 as being very well and having gained weight.

15

1905. 25. 82. 5 lbs. (N. get.)
1 R. II., III.; L. II. (most crackling rales in both lungs).
2 ———
3 Cough for 6 months.
4 X.
5 V.
6 84-100-120 c reactions.
7 F. 99/5 to 100/2.
8 Th. G. 3.
9 Pessily.
10
11 A.
12 Treated with P.T.O., very sensitive, even to small doses (100', 100', etc.) then P.T., also sensitive.
1.12.05. 1002 cc. P.T. 100'.
6.12.05. 1002 cc. P.T. 95'4" (Silent Weight).
9.12.05. 1004 cc. P.T. 100'2".
12.12.05. 1005 cc. P.T. 99'4".
Again:—
5.12.05. 1015 cc. P.T. 100'. Wt. 84. 4 lbs.
12.12.05. 1021 cc. P.T. 103'.
Pulse:—
19.1.06. 1015 cc. P.T. 98'4".
1025 cc. P.T. 100'4".
25.1.06. 1015 cc. P.T. 99'.
30.1.06. 104 cc. P.T. 103'.
Began again with small doses of

P.T.O., and went forward without reaction to 1 cc.; then P.T., with moderate reactions (100'-101') up to 5 cc. Entirely improvement in physical signs, no cough or phlegm, looks well, wt. 92. 8 lbs.

16

1905. 13. 76. 6 lbs.
First child died of tuberculous meningitis; second has had large tuberculous glands of neck removed. Patient's mother, wt. 12. 8 lbs.
1 R. II.; L. II.-III.
2 ———
3 1 year.
4 X.
5 V. Had appetite.
6 90-96.
7 F. 99'4"-100'.
8 Th. G. 3-4.
9
10
11 A.
12 Previous treatment by open abscess wound; treated with P.T. up to 7 cc.
24.1.05. 103 cc. P.T. 104'2". Since severe reaction no cough at all, hardly any phlegm, looks splendid, and appetite good.
In June two reactions of 103'.
In July reactions 100'2"-100'5".
In August reactions 99'2"-101'.
9.1.05. 105 cc. P.T. 100'5".
11.1.05. 7 cc. P.T.
14.1.06. 500 cc. P.T.
Tested 2.06. 1005 Old T., 100, 100, and 103, all negative.
21.06. Went to Melbourne Camp; a terribly wet day; feet wet all day; got a chill and relapsed.
23.11.05. P.T.O. up to 5 cc.; then T.O.A. to 5 cc.; then P.T. up to 125 cc.; seems quite well again; no cough or phlegm and looks quite well on 31.12.1905. In spite of advanced stage of disease (II.-III.) in 1905, 24 years later no symptoms, gain of 15 lbs. in weight, and best of all; tested in December, 1907, with negative result. Such a result demands the confidence and patience of the patient, but it is worth it.
October, 1908. Tested. Did not react to 105 cc. Old T.

17

- diag. 37. cont. ralis.
 No family history. Pl. high in family.
 1 R. II-III; L. II.
 2 —→
 3 3 years. Heart 14 1/2 years.
 Many lesions.
 4 X.
 5 V.
 6 50-80.
 7 N.
 8 To. G. 4. Sputum 1/2 cc.
 9 Haemorrhage.
 10
 11 1/2 cc.
 12 Previous treatment: took arsenate
 for 6 months, did him no good.
 In Queensland for year in treat-
 ment for 6 months, no better for
 it; slowly lost ground; had to
 give up work of any sort.
 Treated with P.T.O. up to 1 cc. on
 Mary reactions (100', 100', 100');
 then T.O.A. up to 1 cc. Then
 P.T. up to 4 cc.
 13. 14. 15. 16. 17. 18. 19. 20. 21. 22. 23. 24. 25. 26. 27. 28. 29. 30. 31. 32. 33. 34. 35. 36. 37. 38. 39. 40. 41. 42. 43. 44. 45. 46. 47. 48. 49. 50. 51. 52. 53. 54. 55. 56. 57. 58. 59. 60. 61. 62. 63. 64. 65. 66. 67. 68. 69. 70. 71. 72. 73. 74. 75. 76. 77. 78. 79. 80. 81. 82. 83. 84. 85. 86. 87. 88. 89. 90. 91. 92. 93. 94. 95. 96. 97. 98. 99. 100.
 101. 102. 103. 104. 105. 106. 107. 108. 109. 110. 111. 112. 113. 114. 115. 116. 117. 118. 119. 120. 121. 122. 123. 124. 125. 126. 127. 128. 129. 130. 131. 132. 133. 134. 135. 136. 137. 138. 139. 140. 141. 142. 143. 144. 145. 146. 147. 148. 149. 150. 151. 152. 153. 154. 155. 156. 157. 158. 159. 160. 161. 162. 163. 164. 165. 166. 167. 168. 169. 170. 171. 172. 173. 174. 175. 176. 177. 178. 179. 180. 181. 182. 183. 184. 185. 186. 187. 188. 189. 190. 191. 192. 193. 194. 195. 196. 197. 198. 199. 200.
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 701. 702. 703. 704. 705. 706. 707. 708. 709. 710. 711. 712. 713. 714. 715. 716. 717. 718. 719. 720. 721. 722. 723. 724. 725. 726. 727. 728. 729. 730. 731. 732. 733. 734. 735. 736. 737. 738. 739. 740. 741. 742. 743. 744. 745. 746. 747. 748. 749. 750. 751. 752. 753. 754. 755. 756. 757. 758. 759. 760. 761. 762. 763. 764. 765. 766. 767. 768. 769. 770. 771. 772. 773. 774. 775. 776. 777. 778. 779. 780. 781. 782. 783. 784. 785. 786. 787. 788. 789. 790. 791. 792. 793. 794. 795. 796. 797. 798. 799. 800.
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 1001. 1002. 1003. 1004. 1005. 1006. 1007. 1008. 1009. 1010. 1011. 1012. 1013. 1014. 1015. 1016. 1017. 1018. 1019. 1020. 1021. 1022. 1023. 1024. 1025. 1026. 1027. 1028. 1029. 1030. 1031. 1032. 1033. 1034. 1035. 1036. 1037. 1038. 1039. 1040. 1041. 1042. 1043. 1044. 1045. 1046. 1047. 1048. 1049. 1050. 1051. 1052. 1053. 1054. 1055. 1056. 1057. 1058. 1059. 1060. 1061. 1062. 1063. 1064. 1065. 1066. 1067. 1068. 1069. 1070. 1071. 1072. 1073. 1074. 1075. 1076. 1077. 1078. 1079. 1080. 1081. 1082. 1083. 1084. 1085. 1086. 1087. 1088. 1089. 1090. 1091. 1092. 1093. 1094. 1095. 1096. 1097. 1098. 1099. 1100.
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 1301. 1302. 1303. 1304. 1305. 1306. 1307. 1308. 1309. 1310. 1311. 1312. 1313. 1314. 1315. 1316. 1317. 1318. 1319. 1320. 1321. 1322. 1323. 1324. 1325. 1326. 1327. 1328. 1329. 1330. 1331. 1332. 1333. 1334. 1335. 1336. 1337. 1338. 1339. 1340. 1341. 1342. 1343. 1344. 1345. 1346. 1347. 1348. 1349. 1350. 1351. 1352. 1353. 1354. 1355. 1356. 1357. 1358. 1359. 1360. 1361. 1362. 1363. 1364. 1365. 1366. 1367. 1368. 1369. 1370. 1371. 1372. 1373. 1374. 1375. 1376. 1377. 1378. 1379. 1380. 1381. 1382. 1383. 1384. 1385. 1386. 1387. 1388. 1389. 1390. 1391. 1392. 1393. 1394. 1395. 1396. 1397. 1398. 1399. 1400.
 1401. 1402. 1403. 1404. 1405. 1406. 1407. 1408. 1409. 1410. 1411. 1412. 1413. 1414. 1415. 1416. 1417. 1418. 1419. 1420. 1421. 1422. 1423. 1424. 1425. 1426. 1427. 1428. 1429. 1430. 1431. 1432. 1433. 1434. 1435. 1436. 1437. 1438. 1439. 1440. 1441. 1442. 1443. 1444. 1445. 1446. 1447. 1448. 1449. 1450. 1451. 1452. 1453. 1454. 1455. 1456. 1457. 1458. 1459. 1460. 1461. 1462. 1463. 1464. 1465. 1466. 1467. 1468. 1469. 1470. 1471. 1472. 1473. 1474. 1475. 1476. 1477. 1478. 1479. 1480. 1481. 1482. 1483. 1484. 1485. 1486. 1487. 1488. 1489. 1490. 1491. 1492. 1493. 1494. 1495. 1496. 1497. 1498. 1499. 1500.
 1501. 1502. 1503. 1504. 1505. 1506. 1507. 1508. 1509. 1510. 1511. 1512. 1513. 1514. 1515. 1516. 1517. 1518. 1519. 1520. 1521. 1522. 1523. 1524. 1525. 1526. 1527. 1528. 1529. 1530. 1531. 1532. 1533. 1534. 1535. 1536. 1537. 1538. 1539. 1540. 1541. 1542. 1543. 1544. 1545. 1546. 1547. 1548. 1549. 1550. 1551. 1552. 1553. 1554. 1555. 1556. 1557. 1558. 1559. 1560. 1561. 1562. 1563. 1564. 1565. 1566. 1567. 1568. 1569. 1570. 1571. 1572. 1573. 1574. 1575. 1576. 1577. 1578. 1579. 1580. 1581. 1582. 1583. 1584. 1585. 1586. 1587. 1588. 1589. 1590. 1591. 1592. 1593. 1594. 1595. 1596. 1597. 1598. 1599. 1600.
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 1701. 1702. 1703. 1704. 1705. 1706. 1707. 1708. 1709. 1710. 1711. 1712. 1713. 1714. 1715. 1716. 1717. 1718. 1719. 1720. 1721. 1722. 1723. 1724. 1725. 1726. 1727. 1728. 1729. 1730. 1731. 1732. 1733. 1734. 1735. 1736. 1737. 1738. 1739. 1740. 1741. 1742. 1743. 1744. 1745. 1746. 1747. 1748. 1749. 1750. 1751. 1752. 1753. 1754. 1755. 1756. 1757. 1758. 1759. 1760. 1761. 1762. 1763. 1764. 1765. 1766. 1767. 1768. 1769. 1770. 1771. 1772. 1773. 1774. 1775. 1776. 1777. 1778. 1779. 1780. 1781. 1782. 1783. 1784. 1785. 1786. 1787. 1788. 1789. 1790. 1791. 1792. 1793. 1794. 1795. 1796. 1797. 1798. 1799. 1800.
 1801. 1802. 1803. 1804. 1805. 1806. 1807. 1808. 1809. 1810. 1811. 1812. 1813. 1814. 1815. 1816. 1817. 1818. 1819. 1820. 1821. 1822. 1823. 1824. 1825. 1826. 1827. 1828. 1829. 1830. 1831. 1832. 1833. 1834. 1835. 1836. 1837. 1838. 1839. 1840. 1841. 1842. 1843. 1844. 1845. 1846. 1847. 1848. 1849. 1850. 1851. 1852. 1853. 1854. 1855. 1856. 1857. 1858. 1859. 1860. 1861. 1862. 1863. 1864. 1865. 1866. 1867. 1868. 1869. 1870. 1871. 1872. 1873. 1874. 1875. 1876. 1877. 1878. 1879. 1880. 1881. 1882. 1883. 1884. 1885. 1886. 1887. 1888. 1889. 1890. 1891. 1892. 1893. 1894. 1895. 1896. 1897. 1898. 1899. 1900.
 1901. 1902. 1903. 1904. 1905. 1906. 1907. 1908. 1909. 1910. 1911. 1912. 1913. 1914. 1915. 1916. 1917. 1918. 1919. 1920. 1921. 1922. 1923. 1924. 1925. 1926. 1927. 1928. 1929. 1930. 1931. 1932. 1933. 1934. 1935. 1936. 1937. 1938. 1939. 1940. 1941. 1942. 1943. 1944. 1945. 1946. 1947. 1948. 1949. 1950. 1951. 1952. 1953. 1954. 1955. 1956. 1957. 1958. 1959. 1960. 1961. 1962. 1963. 1964. 1965. 1966. 1967. 1968. 1969. 1970. 1971. 1972. 1973. 1974. 1975. 1976. 1977. 1978. 1979. 1980. 1981. 1982. 1983. 1984. 1985. 1986. 1987. 1988. 1989. 1990. 1991. 1992. 1993. 1994. 1995. 1996. 1997. 1998. 1999. 2000.
 2001. 2002. 2003. 2004. 2005. 2006. 2007. 2008. 2009. 2010. 2011. 2012. 2013. 2014. 2015. 2016. 2017. 2018. 2019. 2020. 2021. 2022. 2023. 2024. 2025. 2026. 2027. 2028. 2029. 2030. 2031. 2032. 2033. 2034. 2035. 2036. 2037. 2038. 2039. 2040. 2041. 2042. 2043. 2044. 2045. 2046. 2047. 2048. 2049. 2050. 2051. 2052. 2053. 2054. 2055. 2056. 2057. 2058. 2059. 2060. 2061. 2062. 2063. 2064. 2065. 2066. 2067. 2068. 2069. 2070. 2071. 2072. 2073. 2074. 2075. 2076. 2077. 2078. 2079. 2080. 2081. 2082. 2083. 2084. 2085. 2086. 2087. 2088. 2089. 2090. 2091. 2092. 2093. 2094. 2095. 2096. 2097. 2098. 2099. 2100.
 2101. 2102. 2103. 2104. 2105. 2106. 2107. 2108. 2109. 2110. 2111. 2112. 2113. 2114. 2115. 2116. 2117. 2118. 2119. 2120. 2121. 2122. 2123. 2124. 2125. 2126. 2127. 2128. 2129. 2130. 2131. 2132. 2133. 2134. 2135. 2136. 2137. 2138. 2139. 2140. 2141. 2142. 2143. 2144. 2145. 2146. 2147. 2148. 2149. 2150. 2151. 2152. 2153. 2154. 2155. 2156. 2157. 2158. 2159. 2160. 2161. 2162. 2163. 2164. 2165. 2166. 2167. 21

1915a. wt. 112. 24 lbs.; even 112.
24 lbs. Went up country to station,
and looked very ill on return.
Treated again, P.T.O. up to
85 cc., and T.O.A. up to 1 cc.;
then treated with P.T. up to 25 cc.
(103 1/2); pause for six months.

1915b. Very much better; June, 1915,
heard from his brother, a doctor,
that he had gained most weight
and was very well.

1916. Still fairly well, but under
treatment.

21

24. 54, 115 lb.

1 Stage II-III: also only disease of
out.

2 ———

3 3 years.

4 A.

5 B.

6 So.

7 1/2 (100%).

8 Th.

9 Tuberculosis of ankle and mastoid.

10

11 A.

12 Treated for 5 weeks; 2 severe re-
actions; gained 24 lb.; but gave

up treatment because she was a
monthly nurse and could not afford
to give up this work. There
should be a law to prevent this
kind of thing—a nurse, in ad-
vanced consumption, attending
mother and infant. I said so
plainly, and so she went away.

I do not know the sequel, but can
guess it.

22

44. 70-115 lb.

105.

1 Stage II-III.

2 ———

3 1 year.

4 N.

5 V.

6 N.

7 N.

8 Th. G. 5. Sputum 1/2.

9

10 Harbors? tub.

11 A.

12 Treated for 2 months with P.T. up
to 125 cc.

Cough better, has not coughed for
3 nights, much less phlegm; gained
24 lb.; wt. 92. 115.

Left off treatment.

1906

23

74. 80.

106.

Father, 47-55, 84.

1 R. L-II; L-II-III.

2 ———

3 Cough for 5 months.

4 N.

5 V.

6 N.

7 N.

8 Th. G. 9-10. M. micrococci
thousands of colonies on plates.
Sputum 1/2.

9 Hemorrhages.

10

11

12 Treated with P.T.O. up to 75 cc.;
then T.O.A. up to 1 cc.; then
P.T. up to 1 (105 1/2); 80. 24 lb.;
pause for 2 months.

On return (Jan. 06) 105 P.T. 105;
began again with small doses of
P.T.O. Pt. left off coming and

died within 6 months. She had
not had enough tuberculin to do
much good, and she could only die
without it.

24

20.

Th. G. 1-5. Sputum 1/2.

Stage II-III.

Had 4 doses of P.T.O. and then left
off coming.

25

20. 75 lb. 115.

No family history.

1 R. II; L. III; cavity at base
(bronchiectasis).

2 ———

3 2 years.

4 A. Not lost weight.

5 V.

6 N.

- 8 Th. G. 5. Clamps; sputum 1/2.
 9 Pleurisy; laryngitis.
 10
 11 1/2 yr.
 12 Treated with P.T.O. to 25 cc.;
 gained 8 lbs.; wt. 50; cough; then
 P.T. up to 100 cc.; gain; then
 P.T. again up to 15.
 13.06.06. 15 P.T. 100.
 25.02.06. 15 P.T. 101.
 4.07. 15 P.T. 100.
 10.1.07. 175 cc. P.T. 99.
 25.1.07. 225 cc. P.T. 100; up to
 1 P.T. 99.
 11.07. Had severe influenza.
 12.07. T.E. up to 18 cc.; lost wt.
 10 lbs. (11 lbs.); sputum reduced
 from 4 ounces to 1 ounce. In very
 poor condition, can hardly get
 food.
 1909. Wrote last week asking for
 more treatment.

30

- 1.06. 22. 50. 4 lbs.
 1 R. II. L. II.
 2 —
 3 21 years. Hemorrhage, lost a
 quart; second O.
 4 X.
 5 V.
 6 54-90.
 7 N.
 8 Th. G. 3 5.
 9 Pleurisy; hemorrhage.
 10
 11 1/2.
 12 Treated with P.T.O. up to 10 cc.;
 then T.O.A. to 1 cc.; then T.E.
 up to 1 cc.; then T.E. up to 4
 cc. Cough and phlegm ceased.
 Subsequently a small hemorrhage.
 When she came to see she could
 hardly walk a mile. After treat-
 ment she went up and down Federal
 Pass at Katocthon—a good task
 for her.
 March, 1909. Tested; did not react
 to 15 cc. Old T.

31

- 1.06. 27. 50. 4 lbs.
 1 R. II. L. II-III.
 2 —
 3 2 years.
 4 X. Very poor health.
 5 V. No appetite at all.

- 6 90-100.
 7 1/2. 100, 100, 101.
 8 Th. G. 3 5.
 9 Hemorrhage; clamps (pink T.).
 10
 11
 12 Sent to country; no good.
 Treated with P.T.O. up to 10 cc.;
 then P.T. up to 10 cc.; gained
 weight and improved, but I sus-
 pect tubercle (intercostal tuber-
 culum); gained 8 lbs.; weight 90.
 11.07. enlarged and treated again
 with P.T. up to 15 cc.; then
 passed 5 months and again P.T.
 up to 15 cc. (100). From the
 outset I took a grave view of this
 case, and do not hope for any
 good result.
 Really Stage III.
 Died in Feb., 1909.

32

- 1.06. 28. 100. 11 lbs.
 1 R. II. L. II-III.
 2 —
 3 1 year 10 months.
 4 A.
 5 Appetite fair.
 6 50-100.
 7 N.
 8 Th. G. 3 Sputum 2 1/2.
 9
 10
 11 C.
 12 Treated with P.T.O. to 15 cc.;
 then T.O.A. up to 15 cc.; then
 P.T. up to 15 cc. No reaction
 of any account till September, then
 considerably 100. Very great
 improvement; cough much better;
 much less phlegm; looks even at
 much better.
 1909. Know that she is still well.

33

- 1.06. 28. 70. 11 lbs.
 1 R. II-III. L. I-II.
 2 —
 3 Since Jan., 1906.
 4 X. 70 lbs.
 5 V.
 6 54-100-100. Even temp. at 100,
 only increases rate to 106-110.
 7 1/2 and 1/2 (104 and 105) quart from
 (intercostal).

8 Th. G. 4. 50 system.

9 Hysteria for 2 months; low fever.

10

11

12 Previous treatment: Th. G. did no good; sent her to Dr. A. F. in Sydney, who said he "could" do no more, and did not expect her to come back. "Conjugal" is very grave case when I first saw her.

Tuberculin 6 x .06. P.T.O. up to 1 cc., sometimes; T.O.A. up to 5 cc.; then P.T. up to 10 cc. In Oct., 1906 two injections of 100%. Yet much better, and we get along a pair of miles. Went home for first 3 weeks; returned in Spring, 24.1.06. P.T.O. up to 10 cc.; wt. 100 lbs.; very sensitive; up on P.T. 10 cc.; then T.O.A. up to 5 cc.; then T.O. up to 10 cc.; wt. 100 lbs.; then T.P. up to 10 cc.; wt. 100 lbs.; then T.P. up to 10 cc.; wt. 100 lbs.; very sensitive.

Dr. Girdle has reported this case as about *M. G. 1906*, 1907.

March, 1907. She is coming for more treatment.

34

6.06. 40. 100/450

No family history.

1 R. H. - H. L. L. - H.

2

3 1 year. 14 days after operation by Dr. M. G. Simla, had a hemorrhage from lung.

4 A.

5 V. Had fever.

6 96.100.

7 1. 100/450.

8 Th. G. 4. 50 system.

9 Winda 12 months ago.

10-12

12 Hysteria that ever in his life though had fever, has gained no full yet worse. Treated with P.T.O. to 74 cc.; gained wt. 100 lbs. 100/450; then T.P. up to 10 cc.; wt. 100 lbs.; temperature fell 14 mm. degrees to 100.0; P.T. continued up to 10 cc. (200.100). very great improvement, which patient himself admitted, though at the outset he was a skeptic and resistant. He merely came to me because he had

seen the mare that was in Case A. (page 11). Finally, he thought his wife he was to be cured. (1907) Th. G. had no effect upon her.

35

6.06. 40. 100/450

1 Mother 40. 100/450

1 R. H. L. H. - H.

2

3 1 year.

4 100.

5 100.

6 100-100.

7 100-100.

8 Th. G. 4. 50 system 20.

9

10 Positive reflex.

11 100.

12 Treated with P.T.O. to 100 cc.; then T.O.A. up to 74 cc.; then P.T. up to 10 cc.; 100/450. After 14 days treatment the temperature fell 14 mm. degrees from 100.6 to 100.0; then P.T. up to 10 cc. 100. 100. 100.

This was a grave case, and the improvement was greater than I expected; she looked very well.

April, 1907. - Found from Dr. Girdle that she had been to see him, and that it had not improved.

1907. 100/450. 100. 100.

36

6.06. 40. 100/450

1 Mother 40. 100/450

1 R. H. L. H. - H.

2

3 Positive 2 in 1 year ago; very slow; all the year ago hemorrhage 100. 100.

4 100.

5 100.

6 100-100.

7 100-100.

8 Th. G. 4. 50 system 20.

9 Hemorrhage.

10

11

12 Treated with P.T.O. to 100 cc.; then T.O.A. up to 74 cc.; then P.T. up to 10 cc. 100. 100. 100. and temperature gradually fell to

99'. No other treatment. Man was able to come to see from Manly at Sea. Very great im-

provement, much more than I expected or promised. He went back to country. No news since.

1907

37

107. 47. 10st. 7lbs.

No family history.

- 1 R. II.-III. (cavity on basolectasis at base). Good attraction. L. II.
- 2 —
- 3 20 years ago pleurisy.
- 4 N.
- 5 Y.
- 6 90-100.
- 7 100-110.
- 8 Th. G. 4. Sputum 5/11.
- 9 Hemorrhage 10/11.

10

11

- 12 Previous treatment: lock crassid for 1 year; open air treatment at intervals every summer. Tuberculin, P.T.O. up to 3 cc. and P.T. up to 1/4 cc. (10/1/05). Temperature gradually fell to 99'-98' between times; sensitive. For months this patient gave himself the injections into his leg without my trouble after three weeks' instruction by myself. Many other cases in Queensland, New Zealand, Tasmania, and Victoria have been able to do the same without any trouble.

38

107. 42. 10st. 7lbs.

No family history.

- 1 R. II. L. 1-II. Extensive and destructive ulceration of epiglottis, and infiltration of cords; almost no voice.
- 2 Began w. hæmorrhage and pain in throat 7 months ago; sent to Dr. K. Then saw old friend, Dr. McK., a specialist, and nothing could be done, and advised him to go back.
- 3 N.
- 4 R. Good.
- 5 N.
- 6 N.
- 7 N.
- 8 Th. G. 5. Sputum 5/11.

- 9 Ulceration of epiglottis and infiltration of vocal cords; some hæmorrhages.

10

11

- 12 Previous treatment: gained weight, but disease advanced.

Tuberculin, P.T.O. up to 1 cc.; then P.T. up to 5 cc. Extraordinary improvement. Epiglottis healed to all appearance; no cough or phlegm; gained 25 lbs. in 1 yr. Dr. McK. can vouch for these facts. Further, stated with Old T. in Dec., 1906, gave no reaction to 105 cc. Old T. One of the most extraordinary cases in all my experience. He also gave himself most of the doses of P.T. 1909. Still keeping well though I have not seen him.

39

107. 15. 6st. 15lbs.

Always lived in country. Aunt, 14 years ago caught p.

- 1 R. II.-III. L. II.
- 2 —
- 3 Cough since 1906. Pleurisy since April, 1906.
- 4 N.
- 5 B. Appetite good, but patient very pale.
- 6 Pulse good, 80-85.
- 7 N.
- 8 Th. G. 6. Sputum 5/11.
- 9 Pleurisy since April.
- 10 Heartiness for 3 weeks.
- 11
- 12 Treatment in country of no avail.

The patient was brought to me because the doctor held out no hope. Then tuberculin, P.T.O. up to 1 cc. In three months gained 15 lbs. — 5/11; then P.T. up to 1 cc. At this stage (May, 07) a very sharp attack of measles. No injection from May 20 to June 8. Lost weight—21st 15lbs. Then (8/1/07). 105 P.T. 102'; 103 P.T. 99'; 104 P.T. 99'; 105

P.T. 99¹; 1875 cc. P.T. 109¹, up to 1/2 cc. Wt. 90. 11lb. Immense improvement. No cough or phlegm; appetite very good, and gained 15 lbs. It seemed like a miracle. Patient came to see me in Oct., 1906. No symptoms. Tested. No reaction to 1/2 cc. O.T. Her mother had early phos. T. and has just completed course.

40

10.02. 23. 70. 108lb.
 1 R. H. 1. H. 111.
 2 — 3.
 3 0 months.
 4 N.
 5 V.
 6 95-100, 110.
 7 7 and 1/2.
 8 Th. G. 4. System 34.
 9

10
 11
 12 Treated by Dr. H. in Wellington, N.Z., who test first to me that "I might advise her to go to India," etc. Her history was very bad—severe acute attacks with temp. 102¹, 104¹; then chronicity; then relapses. Tuberculin, P.T.O. up to 1/2 cc. in 3 weeks pulse 54-56, wt. 50. 61lb.; P.T.O. up to 1/2 cc., wt. 127lb.; then P.T. up to 1/2 cc. in 3 months gained 20 lbs., wt. 147lb.; P.T. continued to 1/2 cc. (100¹), pulse 78, wt. 90. 61lb.; extraordinary improvement, in 6 months gained 24 lbs., lost cough and phlegm, and looks a fresh, active, healthy woman. I have Dr. H.'s own serious view of her case without tuberculin treatment. Had last week been treated. Patient has never once gone back. Looks and feels well. Wt. 120. 11lb.

Other cases (41, 42, 43, 44, 45, 46, 47, 48, 49, and 50) have been under treatment for but a few months, and I can assert that in every one of these cases, improvement began with tuberculin treatment, and will continue so long as the patients have patience and do their best to help themselves in their dire misfortune. Most of these cases are young girls: 41 *et.* 31, 42 *et.* 23, 43 *et.* 23, 44 *et.* 26, 45 *et.* 17, 47 *et.* 23, 49 *et.* 30, 50 *et.* 19. In all tubercle bacilli were present; but it would serve no useful purpose to do more than say that they are all under tuberculin treatment, and in many of these cases I anticipate a good result. Without tuberculin they would be inevitably doomed—for sanatorium methods and open-air methods do not pretend to be able to do much for such cases. The striking results in these late stages by means of very large doses is surely a complete answer to men who, following Wright, use homoeopathic doses, and to men like Weicker, the great pioneer of the People's Sanatoria, who will, I hope, be encouraged by my experience to try large doses of tuberculin. I was much disappointed with Weicker's latest publication (1908), because one can see that he is still afraid of tuberculin. I think, on the contrary, that tuberculin is the only remedy for all cases

of tuberculosis; if tuberculin fails other methods must also fail. But one must not speak of the failure of tuberculin until it has been tried in large doses. It is not likely that my experience has been singular, or that other factors, such as the climate of Sydney, *which has for years been so heartily endorsed by the medical profession*,¹ have played a great part in the successful results. My results have been so uniform and striking that I can recommend tuberculin in carefully graduated doses up to large doses at proper intervals of time with the utmost confidence. If such results can be obtained in a late stage (Stage II-III.) may one not reasonably hope to cure all early cases of pulmonary tuberculosis with certainty by means of tuberculin? Dr. Weicker describes certain forms of toxæmia which resist treatment. I have rarely seen any cases of this description. They do not form 5 per cent. of the early cases, and in such cases I have shrewdly suspected other conditions, such as miliary tuberculosis, intestinal tuberculosis, genito-urinary tuberculosis, or even extensive tuberculous lesions in glands or special structures (Addison's disease). The Koch's original statement in 1891 is absolutely true, and because the truth was not then recognised, tens of thousands and hundreds of thousands have been sacrificed to the captious and ignorant criticism of Koch's great discovery in 1891.

We now pass to the cases of the Third Stage, when all methods alike must fail in most cases. All admit that when the lung is extensively diseased, and large areas of the lung have softened, and communicate with the air passages, infected ulcerated surfaces result, and mixed infection is the cause of exhaustion and death. Nevertheless even in late stages the energy of tissue may make some sort of fight to protect the individual. Certain it is that even with extensive cavity formation there may be little or no fever and even the pulse may not be fast. One danger is always imminent, the danger of hæmorrhage. Even a large vessel may be exposed on the ulcerated wall, and, being more or less diseased and weakened, may suddenly give way and drown the patient in his own blood. Such an accident may occur even when the

¹ See my article in *Austr. Medical Gazette*.

tuberculous process is not progressing. It may be due to a violent effort; it may suddenly occur at rest or during sleep.

Let me give three illustrations of these severe hemorrhages in the Third Stage and in Stage II. (No. 47).

1

December, 1904

25. 82. III.

- 1 R. II.; L. III. (active)
- 2 \rightarrow
- 3 3 years
- 4 X.
- 5 V.
- 6 110-114.
- 7 P. 100-110 (100-100 b).
- 8 Th. G. 2. Absoluta spina, M.S.
- 9 Hemorrhages.
- 10
- 11 1 to 2.
- 12 Furoin treatment of no avail.

Tubercula T.E. up to 3 cc.; then T.E. up to 100 cc., very severe reaction; then T.E. up to 12 cc.

27.11.04. 17 cc. T.E. 100'4" (wt. 80. 29.04).

24.11.04. 15 cc. T.E. 102'.

10.11.04. 1 cc. T.E. 100'.

24.10.04. 15 cc. T.E. 99'4" (wt. 80.5 lbs.) (very great improvement).

Pass, then T.E. 20 cc.

1.12.04. 2 cc. T.E. 102'4".

8.11.04. 2 cc. T.E. 103'.

27.11.04. 2 cc. T.E. 103'.

Pass again. During this same patient was suddenly called to his gold-shedding work, and on the impulse of the moment made a very heavy physical exertion to put the machinery in order. He brought on severe acute pleurisy. He went to the mountains on his own initiative, and hemorrhage occurred. He went, in fact, to a public sanatorium, where he was "stuffed." No doubt the asthma and the increased amount of blood raised blood pressure, and complicated by the hemorrhage. He returned from the mountains "a perfect wreck," and did not improve. He died from a sudden and severe hemorrhage. But for

his violent physical effort, and his ill-timed visit to the sanatorium in the mountains, he might have lived at least 2 or 3 years. At least so I read from his history.

2

January, 1905

30. 104. III.

- 1 R. II.-III. (active) or (hemibronchitis) and great retention of white cells.
- 2 L. I.-II.

2 \rightarrow

3 2 years

4 X.

5 V.

6 X.

8 Th. 10. 2. Spina 3-4.

9 History. bronchitis, hæmorrhages.

10

11

- 12 Treated with T.E. up to 2 cc.; then T.A. to 100 cc. (hemorrhage stopped); system greatly diminished; wt. 100. 10.04. Tubercula hardly disappeared from system and system almost cured. He got married in 1909. In 1905 he began to lose ground and yet would not take any advice and tampered treatment till 16.10.04 (wt. 90. 6 lbs.); then gave him T.E. up to 15 cc.; passed slow, but gave up treatment. In 1906 he went to a sheep station in the country, and one day, getting excited, shouted out to a stallion hind. A severe hemorrhage occurred, and he died within 48 hours.

Again I say that, if he had taken my advice, he would probably have lived several years longer. Half measures cannot help a man who is in the late stages of the disease.

The third case is No. 47, Stage II. He had had hæmorrhages before treatment, and after full treatment had two severe hæmorrhages, but not followed by any fever. Some days after this hæmorrhage I took another physician at his own wish to examine him, and neither of us could find a single sign of active disease. A hæmorrhage from the lungs in such a case may be post-tuberculous, so that it is conceivable that a man may die of hæmorrhage after he has been cured of pulmonary tuberculosis. There was no reaction such as one would expect from the breaking down of a tuberculous focus; there was merely hæmorrhage from the rupture of a superficial vessel (which might be relatively large) already permanently damaged and weakened by an old and even obsolete tuberculous deposit. This view is further strengthened by the fact that this patient (No. 47, Stage II.) is now (March, 1909) enjoying the best of health. His condition was very serious in May, 1906.

The fourth case of hæmorrhage occurred in:

- M. 20.
 1 L. III.; R. II. Acute pneumonic form.
 2 —→
 3 8 months.
 4 X. Much wasted.
 5 Y.
 6 120—130.
 7 F. Temp. 101°—104°. Continuous.
 8 Tx. G. 5. M. (pneumococci).
 9
 10
 11 5.
 12 I was called in consultation to see

this case. Signs of pneumonic consolidation over whole lung, especially upper lobe. Temp. 105°, 104°. Gave 3 or 4 intravenous injections of collargol (1 c.c.m. of 1%) and the temperature came down to normal, and did not rise again. Nevertheless, lung began to break down. Had a few small doses of P.T.O., but I gave it up, because the case was hopeless. Six months after I first saw him, he had a severe and sudden hæmorrhage, which killed him.

While upon the subject of hæmorrhage I can only repeat what I wrote in 1901, "that so far from hæmorrhage contra-indicating tuberculin, tuberculin is the best means of 'arresting and preventing hæmorrhage.' I have always used tuberculin in cases of hæmorrhages—rather more cautiously—and have never seen a harmful effect; indeed, in every case I have seen the hæmorrhage become less frequent and then stop. I do not remember a case in which hæmorrhage occurred for the first time after tuberculin had been used. Except in the cases given above, I have had

no deaths from hæmorrhage. I am therefore bound to conclude that tuberculin is the very best remedy for hæmorrhage in all stages of pulmonary tuberculosis; if hæmorrhage occurs, it is the fault of the vessel and not the fault of the tuberculin.

Next, I know of one case only in which pneumothorax occurred in any of my cases treated with tuberculin. In this case I find in my notes on physical signs, "signs of very superficial cavity at right apex." This pneumothorax occurred in Prince Alfred Hospital a year after tuberculin treatment, and patient recovered from it—a rare occurrence. Spengler has noted also that the prognosis of pneumothorax is far better in cases in which tuberculin treatment has been adopted.

To return to a review of cases in the third stage. If it is admitted by all that cases in the third stage are "past remedy," some interest attaches to such cases that have been verily snatched from the grave. Even cases in the third stage have to be treated, and I have seen too many of them, because when the methods that are in vogue fail, people are ready to try anything—even the methods of the heretic. I have for some years adopted the practice of refusing to treat these advanced cases until the patients have been already examined by well-known medical men and pronounced to be "hopeless." It must damage a man's reputation to treat cases in the third stage. Even with tuberculin I do not expect to get a satisfactory result in more than 10-20 per cent. of cases. This means of course 80-90 per cent. of failures. Thus for every one who can speak of the benefit derived from the use of tuberculin, there are four to nine people ready to condemn it on their own experience. Only, then, on the understanding that at least two competent men have pronounced the case to be in the third stage, will I make an effort to save the patient from death, at any rate for a few years. It may happen, and it has happened to me, that the doctors do not rightly measure the gravity of the case. Thus one case of severe tuberculous disease of the larynx, which had been treated for a year by a throat specialist, was sent to me, and I

greatly astonished the specialist when I told him that the lungs were in the third stage of the disease. In another case of severe laryngeal tuberculosis, two doctors, one a throat specialist and the other a general practitioner, said that there was not much the matter with the lung when the disease in the larynx was in the third stage. Death in both these cases occurred within four months. *It would have been very easy for these doctors, starting with the idea that the lungs were not very seriously involved, to suppose that the tuberculin hastened the end.* In neither case could tuberculin do more than give temporary relief, and this relief tuberculin can give even in desperate cases.

The cases which I proceed to record have all been pronounced by two—generally three—well-known doctors to be "hopeless."

Three of these cases date back to 1902. They have, therefore, survived six years. One of them got married last year; another is at work in an office and looking the picture of robust health—a case in which tuberculous ulceration of the larynx gradually healed under the influence of tuberculin in large doses; and the third is, in the words of the surgeon who sent him to me as hopeless, a living illustration of the value of tuberculin treatment.

STAGE III.

I.

1102. 17. 9th 5th.
 Fat and breathless, baldy.
 1 R. H. L. I. II.
 2 ———
 3 2 years; had much better from a man who had consumption and died of it a few months later.
 4 N.
 5 V.
 6 Pulse 110—120.
 7 1100°.
 8 T. G. 5.
 9 Lost her vision completely; used to sing well.
 10 Pleurisy; night sweats.
 11 —

- 12 Treated with T.E. up to 2 cc.: very reaction (100°, 104°, 107°, 107°). Lost 4 lbs. in weight; all the better for it. Lost expectoration and recovered energy. Two intravenous doses of T.B. Pulse 100, 72—80; weight, 86 lbs.
 13.04. Then T.E. again up to 2 cc. (100°). 15. Suspected relapse; slight reaction to T.A. Gave P.T.O. 2 cc. and P.T. up to 1 cc.
 Patient looks very well; can sing; has no cough or expectoration. Against my advice she got married at end of 1909. The doctors said she could not live three months in 1902.

2 ✓

DECEMBER, 1901.

36. 400 ghs.
 1 R. III. Signs of early L. I.-II.
 -II.
 2 —
 3 2 years (1) doctor.
 4 X, gut. 6ths.
 5 V.
 6 90.
 7 N.
 8 Th. G. & S. Sputum abundant.
 9 Hemorrhages; obstruction of lumen.
 10
 11 10.
 12 Treated with large doses of Ode T.,
 T.E., P.T.S., T.O.A., and finally
 P.T. up to 5 cc.
 Extraordinary improvement: no
 cough or phlegm; sleep at edge
 of vocal cord spreading out to the
 infundibulum area has healed.
 No sign of active disease in lungs.
 Jan., 1902. In splenic condition
 and back at work; five years after
 weight 121, ghs. Gained 30 ghs.

Control Case.

His sister became affected later;
 tried open air treatment for ten
 months and died a few months
 later in 1905.

In 1904 very remarkable results were obtained in four
 cases:—

4 ✓

36. 210, 1014.
 No family history. "Pigeon breast."
 "Paralytic dist."
 1 R. III. L. II.
 2 —
 3 2 years under Dr. S. S.; obeyed
 all instructions.
 4 X.
 5 V. "Could not sleep," "winded
 in day."
 6 120—125.
 7 100—100 1/2.

3 ✓

NOVEMBER, 1902.

36. 121, 910.
 1 Extensive intercostal of genital-
 urinary tract.
 2 —
 3 A very long history: 25 years.
 Began 25 years ago as tuberculous
 lesion; abscess formed, burst, and
 healed.
 4 X.
 5 V.
 6 N.
 7 N.
 8 Numerous Th. in urine in clumps.
 9
 10
 11
 12 Patient was sent to me by Dr. M.
 after a cystoscopic examination
 in 1902, which revealed abscess
 on surface of bladder; urine on
 settling deposited about 2 inches of
 pus, in which numerous (millions
 of) tubercle bacilli; great dysuria;
 cannot hold water for an hour.
 Treated with T.R. Lungs also
 affected L=II; always moistness;
 100—100 1/2.
 12 100. 2 cc. T.R. 10 3/4 up to
 85 cc. T.R. 100; then T.E. up
 to 100 cc. T.E. 100 1/2.
 1903: four years after, blood urine
 absolutely clear, no albumen, no
 pus; had some periodic trouble,
 causing obstruction. Dr. M.
 opened bladder, and during opera-
 tion saw the scars of the old abscess.
 We. 1 pt. "A living inflammation of
 valve of tuberculous" truly.

- 8 Th. G. & S. Sputum $\frac{1}{2}$ and >.
 9 "Terrible" hemorrhage; severe
 pleurisy.
 10 Voice much affected.
 11
 12 Previous treatment:—2 years ago
 only a cough, no phlegm; took
 plenty of Dr. S. S.'s medicine,
 cod liver oil and salines; saw
 Dr. S. once a week for six months,
 travelling 20 miles each time.
 "Got well"; sent to country;
 "got much worse."

Tolerable.—At first T.E. : very sensitive; 100'5", 100', 100'4", 100'; then P.T.O. up to 1 cc.; gained wt.—84. 6 lbs. increase of 10 lbs. "Not two coughs a day these last three" and a lot of phlegm. "Oh, it was grand!" then T.E. up to 2'2" cc., large dose for boy of 16, yet gained 21 lbs.; wt. 105. 2 lbs. Feb. 30.

One evening, not after a dose, but two days later, was telegraphed for. He had a very extraordinary attack. I found him delirious and violent, noisy and agitated. It was difficult to keep him in bed. He had had no sleep on previous night. Temp. 102°—102°. This attack gradually wore off, and later, in 1900, I visited him and he did not resist.

I have heard of him since and know him to be well.

5

JF. 114. 285.

- 1 R. III. L. II.
- 2 —
- 3 21 years.
- 4 X.
- 5 V.
- 6 88—95.
- 7 2 and 3. 100'5", 101', 100'.
- 8 Th. G. 3 M.S. Much sputum (24).
- 9 Tuberculous laryngitis, cordi cystitis and 101 infiltrates between arytenoids: very hoarse, almost aphonic. Dr. K. said "dilatation of cords." Dr. S. S. ordered "cod liver oil and a quart."

- 10
- 11 1.
- 12 Gave P.T. up to 1 cc. Passed for 4 months; then P.T. up to 75 cc. Passed for 3 months; then P.T. up to 75 cc. 121.100. without any reaction. Gained 2 lbs. Wt. 101. 6 lbs. Appeared a hopeless case at onset, especially with fever and tuberculous laryngitis.

1908. Saw him in excellent health and spirits. No cough at night, and good voice. It is easier to describe such a case than explain it. In 1904 Dr. K. made a point of visiting patient's mother, in London, to tell her that he was dying.

6

28. 90. 285.

- 1 R. III. L. II.
- 2 —
- 3 3 years.
- 4 X.
- 5 V. Very short of breath.
- 6 90, 90, 100; then gradually fell to 72—75.
- 7 100'—100'.
- 8 Th. G. 6. Much sputum (24).
- 9 Old mod. supp. chr. (Tab.) 1900, pleurisy; hæmorrhages 1901, renal abscess; lost wt. at least 30 lbs. 6 lbs.

- 10—11 —
- 12 Previous treatment:—Sent to Australia from India. In New England 4 years; felt well. Then, in Brisbane, got renal abscess and relapse. 1902 at Koroomba. 1903 England. 1904 Macclesfield; came to see end of 1904.

Tolerable.—Treated with P.T.O. up to 15 cc. Two very severe reactions (104'4", 105'5"); then P.T.O. up to 75 cc.; gained 10 lbs.; wt. 101 lb.

In 1906, P.T.O. up to 1 cc.; then T.O.A. up to 1 cc.; then P.T. up to 5 cc. (101'4").

1907. Tested. Reacted to 91 Old T.; then T.E. up to 3 cc.

1908. Had several attacks of pleurisy last year. Is having further treatment.

7

66. 285.

No family history.

- 1 Stage III.
- 2 —
- 3 1 year.
- 4 X.
- 5 V. No appetite.
- 6 80—90.
- 7 N. at subnormal.
- 8 Th. G. 2.
- 9
- 10 Night sweats.
- 11

- 12 Open-air treatment for many months, yet Stage III.

Treated with T.E. up to 75 cc.; then P.T.O. to 75 cc., sensitive; then P.T. to 75 cc., very sensitive; then P.T.O. up to 1 cc. (102', 100', 101'); then T.O.A. up to 75 cc.; then P.T. up to 125 cc.

General and local improvement; diminished spasm; signs of lung drying; appetite much better; in very good spirits; was much depressed; and soon gained 30 wt.: 700-270.

8 ✓

95. 98.
Great cystitis. Under eight doctors; three times in country for "open air." No benefit.

1 Stage III.

2 —→

3 More than 5 years.

4 X.

5 V.

6 90-95.

7 C. 100-105.

8 Th. G. 6. M. Abundant system.

9-11.

12 Trained with T.R. up to 4'2 in. Gained 14 lb.; wt. 101.

Very remarkable improvement; hardly any spasm; "used to be a pickle-bottle full." At this stage his father had an apoplectic fit (cerebral hemorrhage), lay down for several weeks, and then died. Patient being older now had to attend to his father's affairs and gave up treatment. Great shock and great strain caused relapse. Even so, patient did not return to me for nearly a year, and then I told him and his wife that it was too late. He died within six months. This case also illustrates the fatality of half treatment and moderate doses.

1905

In 1905 the two following cases improved immensely, but one went to Orange and the other to New Zealand, and I have had no news of them since:—

8a ✓

11. 211. 1470.
No family history, yet her sister, who has been with her, has been infected. Th.

1 Stage III.

2 —→

3 3 years.

4 X.

5 V.

6 So.

7 N.

8 Th. G. 3. Abundant system.

9-11.

12 Trained with F.T. up to 225 cc. Very great improvement; better appetite, better energy; looks far better and has gained 17 lb.; wt. 84. 1440. Patient had then to go to New Zealand, and I have had no news since.

9 ✓

44. 1000 & 800.
Drooping, wt. 70, 80, and lost with 80.

1 Stage III.

2 —→

3 Cough for 14 years.

4 X.

5 V.

6 N.

7 1-100.

8 Th. G. 2. Spasmodic Th.

9 Ext. obstruction in mouth requiring rubber tooth. (Tuba).

10 Tubercular testis removed 2 years ago, and gonorrheal pleurisy over whole of left side.

11 J.

12 Trained with F.T. up to 15 cc. General health greatly improved; system much diminished & gained 2 lb. Was himself abundantly satisfied with result. No news since he left me.

10 ✓

The most remarkable case of all. Her father, a wealthy man, brought his only and favorite daughter to me when she was in the third stage of the disease. Till then she had been treated for anæmia. I told her father plainly that I could not help him. He implored me to

try. I refused, because the girl could hardly stand, was quite breathless, pulse 120-130, temperature 102°, and the physical signs were extensive and grave over both lungs. The father was importunate, and I agreed to do my best if he first took three doctors from Sydney to consult about her condition and assist truly. The consultations took place at his house, because the girl was too weak and ill to venture out again. The verdict of the three doctors is the highest possible was, "She is dying." I thought no, no, but yielded to the father's urgent entreaties and took her to my private consultation. I gave her three or four intravenous injections of collargol (1 ccm. of 2%), and the temperature slowly subsided. I then gave her tuberculin, and I saw her last December, looking a picture of health, though there was evidence of a slight return.

FEBRUARY.

14. 3rd. Sth.

The notes on this case are as follows:—

- 1 R. II.; L. III.
- 2 ——— (1 leading doctors said "dying.")
- 3 9 months ill; treated for anæmia, and was having "physical culture."
- 4 X.
- 5 V.
- 6 120-128.
- 7 F. and then A.
- 8 Th. G. 7. M.S. = cupful of sputum.
- 9 Hemorrhages.
- 10 Epilepsy.
- 11 — Utterly weak.
- 12 Treated with intravenous injections of collargol, and then P.T. At the end of October, 9 months, temperature fell to normal, and wt. was 70½; a gain of 20½. Further treatment in 1906: P.T. up to 55 cc.; then guinea, and P.T.O. to 75 cc., followed by P.T. up to 225 (June, 1906), and then to 35 cc.; wt. 70½ (Oct.; rate of 20½).
- Oct. 6. Tested. 10025, 1006, 1009, 9967, 1044.

1107. Tested. 104 cc. of Old T.; no reaction.

Yet 4.07. A slight hemorrhage: pulse 100; temp 100°.

Having further treatment.

11

Another case happened enough to disconcert the physicians in attendance. The patient was so weak that she lay in bed and could not even lift her head to feed herself. The doctor told me and her father that she could not live two months. I used tuberculin to prove that even so extreme it does no harm, if properly used.

XII.07.

16. Under 6ft., a skeleton.
- 1 Stage III.
- 2 ———
- 3 1½ years.
- 4 X.
- 5 V. too weak to sit up, a terrible condition.
- 6 110-120.
- 7 F. 107°-104°, later 101°.
- 8
- 9 Intestinal intercatarrh also.
- to C.
- 11 Treated with P.T.O. and intravenous injections of collargol.

After three days temperature came down to 100°, but went up again to 101°; P.T.O. up to 75; then five doses of T.O.A. and P.T. up to 104 cc.

In 4 months the patient was able to get up and do a little work about the house. Before treatment she had not been out of bed for months. At the end of 2 months I told the father I could do no more. He was very grateful for the little I had done. She died 4 months later.

12

18. 3rd. Sth.

Seventy in the house of a popular doctor. At least seven other doctors had seen this case, and they have come round to the view, which I maintained from the first, that she had tuberculosis, bronchopneumonia and pericarditis.

1 Stage III. Complete remission of upper lobe of L. lung, with typical signs of acute (chronic) pneumonia, but lasting in end by 1907, and never continuing for months. The case was considered hopeless by all, but I expressed my willingness to take her into the Prince Alfred Hospital, so that the doctor might be relieved of her.

2 —

3 12 weeks.

4 X. 200-210.

5 Y.

6 100-110.

7 Z.

8 No. Th. found. Vol. cc. 100-110.

9 XL.

10 Abscess in L. arm and back.

11 Z.

12 Treated with P.T.O. and P.T.; very sensitive, but in spite of treatment gained weight and improved. Went up the country, and in 1907 returned to Sydney, and is back again with her old friend "the doctor." Wt. 80; pulse of 90; a record.

The doctor was an utter sceptic—now he is a firm believer—and sent me Case 25 (Stage II-III.), which has also started him.

13

31. 74. 96.

Is an equally inactive and successful case.

Father and mother alive and well. Patient married. Her husband died in 6 months of phthisis. Her two brothers also died. They were all treated by Dr. S. J. Patient had also been treated by Dr. S. J. and Dr. W. N., and was in Stage III. when she came to me.

1 R. III. (L. I-III).

2 —

3 1904.

4 X.

5 Y.

6 60-72. With reaction 100-110. P. 90-110, 100, 102-108.

7 N.

8 Th. G. 7. Much expectoration N.

9 Small hemorrhages.

10-11 Z.

12 Treated with P.T.O. Very sensitive. Many accessory infections (1907, 100-110, 101, 102-103; 11 and 12 months) to reach 25 cc. P.T.O.; pulse 10-10 weeks; then P.T. to 2 cc.

100-110. P.T.O. to 1 cc.; T.O.A. to 7 cc., and P.T. to 7 cc.

Good improvement in every way, hardly any cough, phlegm less than ever. "No phlegm from 2 in the afternoon till morning." "Feel wonderfully stronger."

Dec. 1907. No cough, hardly any phlegm, no blood. Central renal cyst. Dr. S. J.'s results in end of January and December.

14

25. 70. 80.

1 Stage R. II; L. III. Large cavity (very) extending into walls? Hyaline?

2 —

3 Several years.

4 X.

5 Y.

6 N.

7 N.

8 No Th. Profuse expectoration—quite a pint in 24 hours of anarchy case (no liver symptoms or enlargement). No tubercle bacilli to be found, but occasional long chains of streptococci; never seen such long and beautiful chains.

9 Floating.

10 —

11 Z.

12 Before coming to me had been operated on for floating kidney, and in Prince Alfred Hospital had been (suffered) several times by Mr. C. T. B. C. Total in hospital by me 100 cc. T.A. 100. Treated with P.T.O. sensitive; 100, 102, 101, but nevertheless passed to 100, 80, 120, and system greatly diminished; then treated with P.T. up to 1 cc.; phlegm now about 50.

Seen late in 1905. Has been at work for last 2 years; seems very well. Is a very good girl.

15

JANUARY.

32. 796. 1916.
Came to me, because Dr. M. said that surgery would do no more.

- 1 Genito-urinary tuberculosis, cystitis removed and other removed. Urine contained pus, and frequent attacks of inflammation in the ureters—two or three in a fortnight. One attack lasted 10 hours. Since treatment attacks became fewer, and at the end of 10 months he had not had an attack for 7 months.

2 ———

3 Many years.

4 N.

5 Y.

6 N.

7 N.

8 No Th.

9

10

11

- 12 P.T. up to 100; several injections of 100, 200, 300, and one 400, yet he gained weight and improved; then P.T. up to 15 cc.; pause, then P.T.O. up to 15 cc.; T.E. up to 15 cc.; then P.T. up to 6 cc.; then T.E. again up to 15 cc. (18.11.07). Very great improvement, as his own letter tells, and wt. 80. 5/12.

If one can do this in a hopeless case of genito-urinary tuberculosis, what may one do in a relatively early case?

16

NOVEMBER.

31. 1100. 1916. 1883. 1884. 1885.

1 Stage III.

2 ———

3 1 year.

4 N. Fat and breathless. Wt. 111.

5 Th.

6 105-100.

7 N.

8 Th. G. 5. Abundant sputum.

9

10

11

- 12 Treated with P.T.O. up to 15 cc.; then T.O.A.; and then P.T. up to 50; but nearly 2 years, to her own delight and my satisfaction, and at the same time improved beyond recognition.

She lived in Tasmania, 50 miles from a doctor. So I taught her and her mother the use of a syringe, and they together gave her a second course of treatment, chiefly with P.T. up to 75 cc. for 8 months without any trouble. Tested in January, 1907, negative. Tested again by herself in September, 1907, also negative. December, 1907, no cough, no phlegm; wt. 91. 11/12. Almost a miracle.

17

Let me add one more case, to which three doctors can also bear witness, one of them I fear, reluctantly.

60. 12/16.

- 1 R. II.; L. III. Upper lobe absolutely solid.

2 ———

3 1 year.

4 N.

5 V.

6 110-130. Very improved.

7 120-160.

8 Th. G. 6.

9 Fleecy.

10

11

- 12 See below for previous history: tuberculosis treatment: P.T.O. up to 15 cc., occupying 6 months, marked improvement, wt. 105; then P.T. up to 115 cc., two doses of 105 cc., 100 cc.; pause for six weeks; then P.T. up to 125 cc.; wt. 84; then severe intercurrent mixed infection, pneumococcal; temperature, 104.4° for some days; pause; then P.T. again to 145 cc. 100 cc.; wt. 86, 5/16; in all, a price of 200s. for 1907 case.

I have published the details of these hopeless cases treated successfully with tuberculin in large doses in order to show

that in these late stages tuberculin succeeds when all other methods inevitably fail. Now if tuberculin may be used in these "awful" cases—even in large doses—with favourable effects, it would seem obvious that there can be no risk in using large doses in early cases. The one great bogey that was portrayed for us in such vivid and realistic colouring by Virchow was mobilisation of the tubercle bacilli. Virchow started the idea, unfortunately gaining a currency that it little deserved, that tuberculin had the effect of mobilising a sleeping enemy. Virchow's great name, and not the reality of the idea, did more than anything else to damn Koch's magnificent work. Even in the latest stages of the disease I have seen no evidence in favour of this mobilisation of bacilli. It is all moonshine to talk of mobilising the bacilli in the early stages or latent forms of pulmonary tuberculosis. This mobilisation is a common event in pulmonary tuberculosis sooner or later, and the tendency which tuberculin has to cause fibroid changes in the tuberculous formation prevents rather than favours mobilisation. All my experience has convinced me that this idea of Virchow is a mere bogey that need not be considered. Let us give it a decent burial and say no more about it.

It is to me quite a mystery how, after rejecting the method of Koch, the medical profession in England developed a sudden enthusiasm for Wright's method with his opsonic index and his homoeopathic doses. As soon as Wright's work was discussed, I at once challenged the opsonic index as a guide in the graduation of doses. The truth was I had dozens of cases which proved this idea of Wright's to be utterly untenable. Moreover I instructed two men, independently of each other, and both of them well versed in Wright's very clever technique, to test the blood of some of my cases which had been treated by means of large doses and with a total disregard of Wright's method. One of these observers, being very friendly to Wright, excused himself several times from giving me his results. He found the opsonic index *so high* that he thought he must have made a mistake. The other man supplied me with these startling opsonic indices: 2.3, 1.78, 1.57, 1.6, 1.3, 1.2, and in no single

instance was the opsonic index under 1. Accordingly, by disregarding the negative phase and proceeding on my own plan in contravention of all Wright's ideas and methods I arrived at results which were proved by Wright's own methods to be infinitely better than his own. In fact, I hoisted my old friend Sir A. E. Wright with his own petard. Would that the wave of enthusiasm that ushered in Wright's work in 1904 had been reserved for a better cause. Sir A. E. Wright cannot be ignorant of the work that I was toiling over long before he thought of entering the field; and although I know that my method is infinitely better than his, I had to labour conscientiously and patiently for nearly seven years before I published anything, and when I did publish there was nobody to take any notice of my work. From my standpoint at any rate I am bold enough to assert, that even in very late stages large doses of tuberculin may do a marvellous amount of good, while nothing else does any good at all; why then should large doses do harm in the earlier stages? *A priori* there is no reason to expect harm, and *a posteriori* I have proved in hundreds of cases that large doses do an extraordinary amount of good, far more good than any other known method.

Surely, too, my experience is only in harmony with the results of experiments. The aim of tuberculin treatment is to stimulate and increase the vigour and amount of the antibodies or weapons of defence evolved by the activity of living tissue, and the more perfect the response to this antigen, tuberculin, the better the mechanism of immunity. Surely the obvious inference is that progressive and relatively large doses will develop a more perfect and more responsive mechanism of immunity than homoeopathic doses of the same strength repeated at long intervals of time. Again let experience teach. The almost invariable effect of increasing doses of tuberculin has been general improvement and abatement of the symptoms. I do not deny that much skill and experience may be necessary to graduate the doses, but if we are aiming at a distant goal, we cannot expect to reach it by marking time or standing still. If anyone, who has tried the method of small doses for a number of years, has been

able after a long interval of time—certainly not less than three or four years—to look back upon the results with exhilarating satisfaction, then he is justified in using and advocating small doses. I can look back over a number of years with great satisfaction upon results that have been achieved with large doses, and shall continue to use large doses.

PERCENTAGES IN FULLY TREATED CASES

1902

Results in 1908 :—

- Stage I. 2 out of 3 treated alive and well; the other one went to New Guinea and died of severe fever (malaria); he had a double larynx infection while I was treating him.
- Stage II. 4 cases: all alive; 2 of these cases quite well for five years, but now having further treatment.
- Stage II.-III. 1 case quite well; 1 case no news lately; 1 case died in 1907.
- Stage III. 6 cases: 3 alive and wonderfully well; 3 died within a year of treatment; all hopeless from the outset.

Percentage results after 6 years :—

- Stage I. 100 per cent.
- Stage II. 50 per cent. well.
- Stage II.-III. 50 per cent. well.
- Stage III. 50 per cent. well.

1903

Results in 1908 :—

- Stage I. 14 cases: 2 died of abdominal operation; 2 were well in 1906; 11 remaining alive and well.
- Stage II. 3 cases: 1 fully treated, quite well; 1 partially treated, lost sight of; 1 who gave up treatment when a few doses, died in 1904.
- Stage II.-III. 4 cases: 2 quite well; 1 gave way to drink and relapsed.
- Stage III. 2 cases not treated by tuberculin died—one refused treatment when in Stage I; other was sister of Case 2, Stage III; he had tuberculosis and is wonderfully well; she had no tuberculosis and died two years ago; 1 case died of haemorrhage.

Percentage results after 5 years :—

- Stage I. 100 per cent.
- Stage II. 100 per cent. (1 cases fully treated).
- Stage II.-III. 60 per cent. (the other case should be well but for potus).
- Stage III. 0 per cent.

1904

Results in 1908 :—

- Stage I. 13 cases: all alive so far as is known: 1 case lost sight of since 1906; other 12 alive and well.
- Stage II. 8 had full treatment and all well; 3 had partial treatment, improved and let off; 1 refused tuberculin treatment and went to Australia—he is dead.
- Stage II.-III. 5 cases: 3 fully treated, very well; 1 partially treated, alive; 1 case left with not heard of.
- Stage III. 7 hopeless cases, died within 6 months; 1 case lost sight of; 4 cases fully treated, marvellously better.

Percentage of *fully* treated cases.

Stage I. 100 per cent.

Stage II. 100 per cent.

Stage II.-III. 100 per cent.

Stage III. All cases fully treated, marvellously better.

1905

Results in 1908 :—

Stage I. 47 cases : 4 lost sight of ; other 23 cases well.

Stage II. 15 cases : 1 case refused treatment and died within a year ; 4 fully treated, known to be well ; 4 gave up treatment after a few doses.

Stage II.-III. 50 cases : 1 had 2 months' treatment only and died in a year ; 1 had two doses, not heard of ; 8 other cases marvellously well.

Stage III. 40 cases : 2 cases quite hopeless, some improved but history unknown ; 7 were too bad for treatment ; 6 fully-treated cases are extraordinarily well.

Percentages after 3 years :—

Stage I. 100 per cent., so far as it known.

Stage II. 100 per cent., fully treated ; the only case known to be dead refused treatment.

Stage II.-III. 8 out of 10 were fully treated ; 100 per cent.

Stage III. 6 out of 14 cases very well ; 45 per cent.

1906

Results in 1908 :—

Stage I. 24 cases : all well.

Stage II. 24 cases : not a single death ; 22 known to be well.

Stage II.-III. 15 cases : 8 known to be well ; 4 left off treatment ; 1 not at all well ; 2 partially treated, died.

Stage III. 25 cases : 11 died within 6 months, having had little or no treatment ; 7 cases more or less treated, improved ; others lost sight of.

Percentage results in 1908 :—

Stage I. 100 per cent.

Stage II. 90 per cent.

Stage II.-III. 50 per cent.

Stage III. 20 per cent improved.

AFTER RECORDS

SUMMARY OF RESULTS OBSERVED IN 1907-1908

Cases treated in 1902:—

- Stage I. 7 cases: 1 man went to New Guinea and died of malaria; no deaths known; 5 cases known to be well.
 Stage II. 4 cases: all alive; 2 had relapse after 5 years, but are improving again.
 Stage II-III. 2 cases: 1 quite well at school; 1 lost sight of; 1 very late, died in 1907.
 Stage III. 5 cases: 3 marvellously well; 2 died within a year, hopeless from the start.

Treated in 1903:—

- Stage I. 14 cases: 1 died of abdominal operation 1907; 1 partially treated, lost sight of; 11 known to be well in 1907; 1 well in 1906, not heard of since.
 Stage II. 1 case: 1 gave up treatment after few doses, he died within a year; this is "control" case; another went away after 5 weeks, lost sight of; 1 known to be quite well in 1906.
 Stage II-III. 3 cases: 2 remarkably well in 1908; 1 improved immensely and then gave way to drink, yet alive in 1907.
 Stage III. 1 case: improved immensely under treatment, but through holiday art brought on hæmorrhage and died.
Control case.—In 1903 had two "control" cases. One was tested by me in 1906, was then in Stage I. She refused tuberculosis treatment, and in 1905 was in Stage III., hopeless; another had antituberculous treatment for a year and was dying.

Treated in 1904:—

- Stage I. 13 cases: all alive and well.
 Stage II. 10 cases: 2 left off treatment, could not afford time; 1 partially treated, great improvement, left off coming. Remaining 7 well.
Control case.—1 "control" case. This patient decided at first to have treatment. Then pretended to go to sanatorium. Was excluded in 1905 as successful case of anaemia, and was dead in two years and a half.
 Stage II-III. 5 cases: 2 left off treatment; other 3 surprisingly well in 1908.
 Stage III. 14 cases: 9 hopeless cases, died within a year; 2 cases improved extraordinarily; 2 other cases were alive in 1908; 1 case improved greatly, but lost sight of since 1906.

Treated in 1905:—

- Stage I. 27 cases: 22 had full treatment, and all are known to be well in 1908 except one, who died of splenic anaemia in 1906; 5 had partial treatment.
Control case.—M. 30, Stage I., refused treatment by me, "because she saw another doctor, who did not feel tubercle in sputum." In 1907 Stage III.

Stage II. 12 cases; 3 had only few doses; 8 had full treatment, and were well in 1907.

Control case.—1 "control" case: put off treatment because he was too busy; he died within a year.

Stage II-III. 9 cases; 8 improved immensely, and were well in 1908.

Control case.—1 gave up treatment because he had no time; he died within 18 months—4 further "control" cases.

Stage III. 20 cases; 9 hopeless cases, could not take treatment, died within 18 months; 7 cases are alive, marvellously better; 4 cases temporarily improved.

Treated in 1906:—

Stage I. 25 cases; 24 fully treated and well; 1 partially treated.

Stage II. 26 cases; 22 cases well; 4 cases partially treated.

Stage II-III. 14 cases; 2 deaths before completion of course of treatment; 1 improved but left off; 1 not benefited; 8 cases very well.

Stage III. 22 cases; 15 cases hopeless, and died within 6 months; 7 improved very much, two of them to an extraordinary degree.

Treated in 1907:—

Stage I. 34 cases; 23 cases fully treated, all better or well; 7 partially treated; 2 no longer treatment.

Stage II. 41 cases; 10 had treatment, and are better or well; 9 with under treatment; 2 gave up after a few weeks' treatment.

Stage II-III. 14 cases; all improved, 1 fully treated are surprisingly well; others under treatment.

Stage III. 12 cases; 9 hopeless; 3 improved.

These records bear out the conclusions I had formed in 1902, that in Stage I, tuberculin secures a successful result in almost 100 per cent. of the cases, certainly in 90 per cent. of the cases, *controlled by after examinations even five years after treatment.* Even in Stage II, my results with tuberculin show successful treatment in fully 60 per cent. to 70 per cent. of the cases. According to my experience it is quite exceptional to meet with a case in Stage II, which cannot be arrested for many years, and it is my opinion, based upon experience, that if these cases are subsequently examined once a year by means of tuberculin, and, if reacting, treated further, the disease may be held in check, and perhaps even permanently arrested. With regard to the later stages, I have been surprised beyond measure at the many successes I have obtained in Stages II-III, and even in Stage III.

Indeed, to any one who has had my experience, it appears clear that the results claimed by Modler and Weicker,

Bandelier and others, for the combined methods of the sanatorium and tuberculin treatment, are in effect the *essence of tuberculin treatment*. Accordingly, if a scheme for the exploitation of tuberculin treatment among the poor be substituted for sanatorium treatment, we shall be acting in obedience to the direct teaching of science and experience. As I have already indicated, *the ideal method of exploiting tuberculin treatment among the poor is by means of tuberculin dispensaries for consumption instead of sanatoria.*

FURTHER CASES TREATED IN 1906.

1

1905. 13. 8m. 12 1/2 lbs.

- 1 R. I.-II.; also L. L.
 2 —
 3 5 years.
 4 A. Severe abdominal pain.
 5 V. Indigestion, varied pain.
 6 Bg.—con.
 7 F (bow).
 8 No TH. Tenes, frequent, after and
 undressing. 10/10 P.T.O. 101 1/2.
 9 Scar in neck (abscesses?)
 10 —
 11 1.
 12 Treated with salicylate. P.T.O.
 up to 95 cc.; reactions, 100 1/2,
 100, 100 1/2; then P.T. up to
 9 cc. (90 1/2).
 Has improved very much, and pains
 in abdomen are less frequent and
 less severe.
 Large ur. pipe; passed 9/10.

2

1907-1908. 44. 100.

- 1 R. H., I.-II.; many sides; late
 second stage.
 2 —
 3 45 months.
 4 A.
 5 B.
 6 S.
 7 N.
 8 Tn. G. 4-5. 3.
 9 Hemorrhoids.
 10 —
 11 —
 12 Treated with salicylate. P.T.O.
 up to 1 cc.; early reactions at
 100, also 101, 102, 104.
 Then P.T.
 15.10.08. 95 cc. P.T. 102.
 20.10.08. 99 cc. P.T. 101.
 26.10.08. 12 cc. P.T. 100 1/2.
 At this stage, in spite of treatment,
 "patient says he feels much."
 He has no sign of disease.
 P.T. continued.
 There was a break here because
 supply of P.T. temporarily failed.

- 16.10.08. 12 cc. P.T. 99 1/2.
 25.10.08. 18 cc. P.T. 100.
 4.11.08. 2 cc. P.T. 99 1/2.
 10.11.08. 25 cc. P.T. 99.
 12.11.08. 35 cc. P.T. 100.
 15.11.08. 4 cc. P.T. 100.
 2.12.08. 5 cc. P.T. 100.
 14.12.08. 6 cc. P.T. 101 1/2. Very
 well.
 27.12.08. 6 cc. P.T. 101.
 3.1.09.09. 7 cc. P.T. 100 (very little
 in system since reaction).
 10.1.09. 2 cc. (special) no weight
 105 (105) gained; no night sweats
 since onset of attack of disease; 1
 kidney stone.
 16.02.09. Treated to 105 cc.
 He is in here a further course of
 salicylate treatment, concluded
 with Spengler's Immune Serum
 (L.S.). Gained wt. 14 1/2 lbs.

3

1907-1908. 41. 90. 8 1/2 lbs.

- 1 R. H. or H.-II. L. I.
 2 —
 3 5 months; weight 8 1/2 lbs.
 4 —
 5 —
 6 —
 7 —
 8 Tn. G. 3-4.
 9 —
 10 On. atrophic keratosis.
 11 —
 12 Treated with salicylate, 20.07;
 P.T.O. up to 95 cc.; wt. 96.
 7/10. — then P.T. up to 525 cc.,
 wt. 96; 2 1/2 lbs.; reaction away,
 99 1/2, 100, not more; then P.T.
 up to 225 cc.; wt. 97. 9/10.
 11.08. — Feels very well.
 After course of Bism. treatment—
 Hb. 95%; neutrophils, 65%;
 lymphocytes, 31%; eosinophils,
 5%.
 Arsenic's table:—
- | 1 | 2 | 3 | 4 | 5 |
|-------|-----|-----|-------|----|
| 11.5% | 41% | 28% | 15.5% | 6% |

10. vii. 08. 725 cc. P.T. 100 V.
 19. vii. 08. 745 cc. P.T. 99 V.
 28. vii. 08. 74 cc. P.T. 100 V.
 7. ix. 08. 445 cc. P.T. 100 V.
 16. ix. 08. 475 cc. P.T. 100 V.
 25. ix. 08. 35 cc. P.T. 100 V.
 4. x. 08. 65 cc. P.T. 100 V.
 18. x. 08. 85 cc. P.T. 100 V.
 5 cc. I. reaction.
 375 cc. J. reaction.

Suspended treatment on account of reaction.

March, 1909, very well. Will be tested in April. (Note an unexpected improvement in Stage III. Later I shall try a combination of tuberculin and Spengler's immune bodies (L.K.), as I fully expect a reaction.)

8

1908. 29. Oct. 12 lbs.

Set in Stage II-III. (No. 7, just preceding case.)

- 1 R. I. L. I.
 2 —→
 3 12 months.
 4 A.
 5 B.
 6 —
 7 —
 8 No Th. 401 cc. (Old T. 102', lasting 24 hours.
 9 Spitting up blood for 22 months.

10
 11

- 12 Treated with tuberculin: P.T.O. up to 1 cc.; and then P.T. up to 8 cc. Wt. rose 6 lbs.

4. x. 08. Feels very well. No spitting of blood for 2 months; but 1 day's work during the whole course of treatment. Rales have disappeared.

In early stage; reaction had not settled much. Gained 12 lbs.

9

1907-1908. 19. Oct. 12 lbs.

- 1 R. II. L. II. Rales and frictions.
 2 —→
 3 Less than 3 months.
 4 X.
 5 V. Appetite poor—baked very ill.

- 6 80-90.
 7 F. 100', 101'—101'.
 8 Th. G. 2-3 in clamps.
 9 Small hemorrhages and haemorrhoids.
 10
 11

- 12 Treated with tuberculin: P.T.O. up to 7 cc. Oct. 20 (116)—gain of 12 lbs. Few reactions, 800', 100 V.; then P.T., much more sensitive, 102', 102', 102' (108 P.T.); then five doses of T.E.; then P.T., no reaction till 3 cc. (100 V.). Then reactions, 375 cc. 325 cc. 102' = 105 cc. 102'; 345 cc. 101'. Flare for 3 weeks.

1. vii. 08. 75 cc. P.T. 99 V.; gained 7 lbs.

10. vii. 08. 2 cc. P.T. 102'.
 19. vii. 08. 2 cc. P.T. 99 V.
 28. vii. 08. 25 cc. P.T. 101 V.
 5. vii. 08. 25 cc. P.T. 106 V.
 11. vii. 08. 3 cc. P.T. 100 V.
 21. vii. 08. 35 cc. P.T. 100 V.
 7. x. 08. 45 cc. P.T. 102'.
 12. x. 08. 45 cc. P.T. 102'. Oct. 12 lbs.

25. x. 08. 45 cc. P.T. 100 V.
 Treatment suspended for a time.

1909. Very great improvement in spite of severe reactions. Gained 7 lbs.; very good considering stage and size of patient—very small.

10

1907-1908. 19. Oct. 12 lbs.

- 1 R. II. III. = III. L. II. c. Tuberculosis laryngitis.

- 2 —→
 3 Cough 2 years—worse in evening.
 4 X.

- 5 V.
 6 60-100.

- 7 F. 101', 101 V.
 8 Tuberculin bacilli G. 3-3. 85.

- 9 Hemorrhages (G. and 3gt.) in February, 1909; and tuberculosis laryngitis.

- 10 Rheumatic pain.

11 —

- 12 Under Dr. W. for 1 year and 4 months. Open air, 7 months in country. Sputum not examined and temperature not taken. Then tuberculin treatment, when patient in Stage III.

P.T.O. up to 1 cc., many reactions.

(100', 100' often); then P.T. up to 5 cc. (100 §').

Progress slow at first, and almost every dose of P.T. caused reaction, but still there was gradual improvement, and in September, 1908, after 10 months' continuous treatment, very great improvement, and weight 90, 15½; right chest still pink and edge rough—and pulse 60 to 65 to 70 to 80, never 100; temperature also normal for weeks.

March, 1909. Tested. Rejected. Treating now with P.T. and Spengler's *vacuum filter* (L.K.). Gained 14½ lb., even though in Stage III.

11

1908. 20. 1909.

Case 106. Stage I.

Father and mother 40.

1 R. L.-H. Killen. L. 1).

2 —→

3 Cough 3 months.

4 A.

5 B.

6 N.

7 N.

8 Phlegm, 3½ yellow. No Tl. (tubercle bacilli). Tested: 100 cc., T.A. 984'; 105 cc., T.A. 121'—headache; cough worse.

9

10

11 A.

12 Treated with tuberculin (P.T.O. up to 9 cc.—no reaction; rest. Gls. P.T. up to 7 cc.—rest. Gls. Three weeks later 100 cc. 11½; felt quite well. Often pain in epigastrium after meals. Gained 14½.

12

1908. 22. 1909.

After phlegm, passed tubercle bacilli in Australian Mineral Print. Society.

1 R. H. L. I.

2 —→

1 up to 100 cc. Gls. died of phlegm.

2 I have never before seen such reaction, though when the skin has peeled off after a contraction.

3 October, 1907, influenza (3 months); phlegm 11 years ago.

4 A.

5 B.

6 > 72-80.

7 N.

8 Tl. G. 3-4 (many tubercle bacilli).

9

10 1½. O.H. advised to sell home, give up position, and go to country; "only hope."

11

12 Treated with tuberculin (P.T.O. up to 1 cc.

2.12.08. 1 cc. P.T.O. 954'.

3.12.08. 10 cc. P.T. 102'.

11.12.08. 6½ cc. P.T. 98'.

10.12.08. 10 cc. P.T. 100'.

17.12.08. 8½ cc. P.T. 101'.

24.12.08. 10 cc. P.T. 984'.

No further reaction with large doses for 6 weeks, and—

17.01.09. 8 cc. P.T. 100'; rest. Gls.

21.1.09. Very well; rest. Gls.

23.1.09. States that he did not lose half a day's work during whole course; now he has no cough and no phlegm—feels "like a champion," "better than sitting home and going to country to waste." Gained 4½ lb.

13

1908. 25. 1909. 11½.

Bother has 0.

1 R. I.

2 —→

3 3 mm. 10.

4 A.

5 B.

6 74.

7 N.

8 No Fl. Rejected, 100 cc., 984'; arm very sore. 100 cc., 998'; arm dreadfully sore and swollen, could hardly move hand. Severe headache.

9

10

11

12 Treated—very sensitive.

14.08. '04 cc. P.T.O. 100'2. Reactions with every dose 99'8—100'7.

15.vi.08. '04 cc. P.T.O. 102'.

18.vi.08. '04 cc. P.T.O. 98'2.

18.vi.08. '05 cc. P.T.O. 101'.

No further reaction. The P.T.O.

increased to 1 cc., then P.T.—

8.viii.08. '25 cc. P.T. 100'4.

15.xi.08. '25 cc. P.T. 101'4.

21.xi.08. '25 cc. P.T. 99'.

No further reaction, and P.T. in-

creased to '35 cc.

21.12.08. '35 cc. P.T. 99'. Patient

very well.

This case shows how doses may be

increased to maximum in spite of

marked dose-sensitiveness—no

answering to Wright's system.

Very good weight at start, because

disease in early stage. Would not

wish her to gain weight; better if

she lost a little.

14

1908. 67. 9st. 10½lb.

(12-41 more 4 years ago)

1 R. H. L. H.-III.

2 —→

3 2 months ago treated for catarrh by

W., who could see bleeding from

throat. Probably 2 years ago

(influenza).

4 X.

5 N.

6 90.

7 99'—99'6'.

8 Th. G. 5-6. Sputum yellow; 3)

blood.

9 Hemorrhage—not large.

10

11

12 Treated for catarrh. Tuberculin,

P.T.O. in small doses.

18.x.08. '006 cc. P.T.O. 102'8.

Several mild reactions, 99'6',

99'5'.

vi.11.08. '23 cc. P.T.O. N. and

normal between doses.

28.viii.08. '1 cc. P.T.O. N. 9st.

11½lb.

Then P.T.—

22.08. 100st. 9st.; gained 4½lb.

28.08. '7 cc. P.T.

Very great improvement—sneezes;

cough much better and less

phlegm. Nocturnal *Tremor*

for months at intervals (see); will

need more treatment. Have pre-

sented further treatment with

Spongel's increasing doses (L.K.)

15

1908. 16. 7st. 10½lb.

One is other Ap.

1 R. H. Early.

2

3 Influenza 3 months ago.

4 N.

5 R.

6 (116-120) (small) (syndromal) and

(continued) pulmonary (second

round).

7 N.

8 Th. G. 2.

9 Small hemorrhage.

10

11

12 Treated with tuberculin: P.T.O.

up to 1 cc. 7st. 12½lb. Then

P.T. up to '325 cc.

10.vi.08. '325 cc. P.T. 104'.

22.vi.08. '325 cc. P.T. 100'.

1.viii.08. '4 cc. P.T. 101'. 7st.

12½lb.

18.vi.08. '2 cc. P.T. 101'.

22.vi.08. '33 cc. P.T. 102'.

Patient for 5 weeks—then began with

'1 cc. P.T. and increased at in-

tervals of 2 days up to '33 cc.

(Oct. 28, 1907) W. 8st.—8st.

4st.

11.08. No phlegm at all. W. 8st.;

gained 4½lb.

16

11.1908. 27. 8st. 9½lb.

(never above 7st.).

1 R. H. L. H.-III.

2

3 2 months.

4

5

6

7

8 Th. G. 3-4. Phlegm yellow; 8st.

9 Laryngeal T. (swelling of aryten-

oids).

10

11

12 Previous treatment, in May hemor-

rhage—sent by W. H. to Hobart,

got worse—lost weight and came

back. Saw Dr. H., who sent him to me.

Treatment with tuberculin: P.T.O. up to 1 cc.; then P.T. up to .5 cc. Wt. lost 4 lbs. (Oct., 1908). Temp. at start 99°—100°, and at end of treatment 99°. Very great improvement in every way considering the stage.

11, 1909. Will need more treatment in 6 months.

Gained 4 lbs. Small person (64).

17

1908.

105. 89.

Hospital case—Stage I.

Treated with P.T.O. to 1 cc.; then with P.T. up to .7 cc. Wt. lost 2½ lbs. Feels splendid, no pain. Gained 5 lbs. (24) lbs.

18

10. lost 4 lbs.

Case already reported, No. 107, Stage I, and continued in 1908.

Three similar doses, 100', 101', 102'4'. Therefore treatment repeated for 3 months. Then, 1, 1, 08. P.T.O. up to 1 cc.; in 23 days, no reaction. Then P.T. up to .264 cc. without reaction, showing greater tolerance after previous P.T. continued—

25, vii, 08. 37 cc. P.T. 100'. 114. 9½ lbs.

23, vii, 08. 39 cc. P.T. 100'.

31, vii, 08. 46 cc. P.T. 100'.

2, vii, 08. 33 cc. P.T. 100'.

17, vii, 08. 5 cc. P.T. 102'4'. 114. 3½ lbs.

27, vii, 08. 6 cc. P.T. 100'4'.

7, ix, 08. 6 cc. P.T. 100'4'.

26, ix, 08. 7 cc. P.T. 99'6'. 114. 6½ lbs.

22, ix, 08. 8 cc. P.T. 99'6'.

5, x, 08. 35 cc. P.T. 100'.

Nov. 11th, 1908, 11th, 11th.

This shows that by increasing the doses greater tolerance is induced, and the climatic result was highly satisfactory, hardly any phlegm and only slight cough in morning; looks and feels very well.

Gave 1 1/2 lbs.

19

25. lost 1 lb.

See Case 107, Stage I.

Treatment continued in 1908.

P.T.O. up to .9 cc.; no reaction. 1st 8½ lbs. Then P.T.: no reaction till—

10, i, 08. 95 cc. P.T. 102'4'. 110. 6½ lbs.

20, i, 08. 95 cc. P.T. 99'4'.

Had intercurrent attack of vomiting, and severe diarrhea; lost weight—lost 1½ lbs.

P.T. was subsequently continued up to .9 cc.

1, viii, 08. 5 cc. P.T.: no temp. Wt. lost 10½ lbs.; gained 4½ lbs.

March, 1909.

I have, besides, thirty-two more cases under treatment, all of which, with one exception, have improved under tuberculin—but such temporary improvements are of no value in helping one to form an opinion upon the benefit of the specific system of treatment.

SERIES OF CHARTS

The following series of charts, giving doses and resulting temperatures, show that even in late stages (Stages II-III and III) *excellent results can be obtained by large doses, and by decreasing the doses even in spite of reactions.* Not seldom progressively increased doses (within reason) finally fail to cause any reaction at all—*altogether against Wright's theory of the "negative phase."* By this system the symptoms gradually lessen, the cough abates, the sputum diminishes in quantity and improves in character till it ceases altogether. Appetite improves, the pulse improves, and the patients are conscious of a gradual recovery of strength and energy. The weight, too, increases uniformly, and often the weight reaches a point never reached before. This improvement occurs even though the patients have lived near Sydney the whole time, and beyond general directions with regard to food and the necessity of avoiding over-exertion and excitement, have not been placed under the strict sanatorium system. Accordingly tuberculin has a very powerful effect in restoring and promoting healthy tissue-metabolism in the manner already carefully described by Mitsuescu of Bucharest in his laborious and comprehensive analysis of the excretions in cases treated with tuberculin (Nitrogenous Metabolism).

Lastly, I have had several blood counts made, in some cases under treatment, *according to Arxell's tables.* These investigations show that by using even large doses of tuberculin the character of the polynuclear neutrophile leucocytes gradually improves. Before treatment the mononuclear neutrophile leucocytes constitute from 25 to 30 per cent., the leucocytes with two nuclei and three nuclei mainly make up the rest. Leucocytes with four and more nuclei practically fail. After treatment, not immediately, but some time after, the blood tables gradually return to the normal. In the normal table, roughly speaking, there are about 3 per cent. of leucocytes with one nucleus, 35 per cent. of leucocytes with two nuclei, 41 per cent. with three nuclei, 17 per cent. with four nuclei,

and 2 per cent. with more nuclei. It is clear that the latter varieties are the more mature and therefore the better trained to defend the body against the attack of organisms.

This improvement in the defensive apparatus of the blood is further confirmatory evidence of the value of large doses of tuberculin in the treatment of pulmonary tuberculosis.

These blood counts involve much time and labour, and I am grateful to Dr. Griffiths for having made most of these investigations at my instigation. I am convinced that Arneth's investigations are a valuable addition to our methods of controlling our results. Personally I consider Arneth's system of blood examinations of more practical use and more trustworthy, though perhaps not less laborious, than the fluctuating and capricious opsonic index of Wright—at any rate in pulmonary tuberculosis.

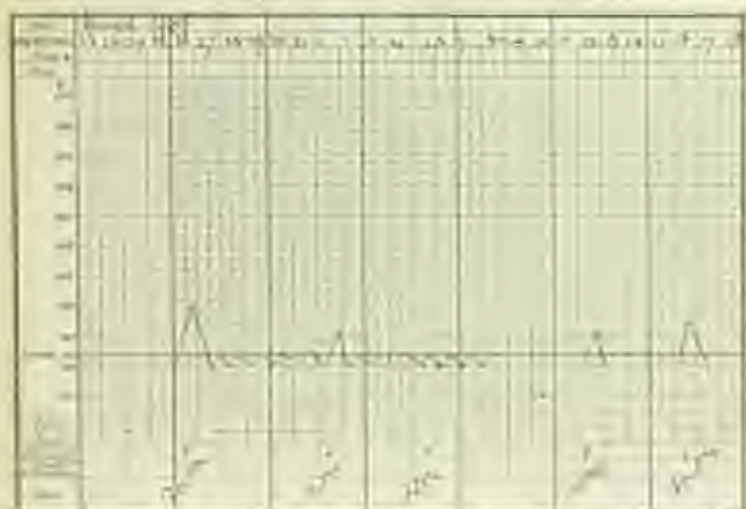
Appended are (1) the records of doses and temperatures already referred to; and (2) tables of blood count in accordance with Arneth's system.

NO. 40. STARK II.



V-4

1000



No. 22 STAGE II.

1

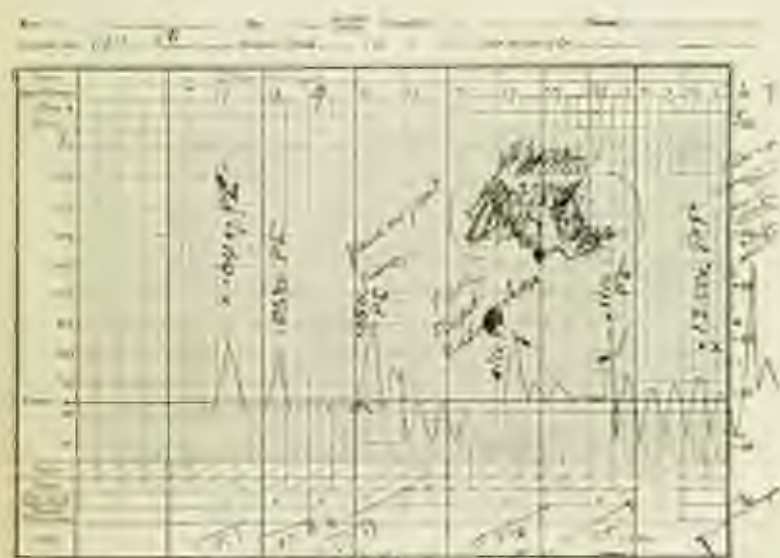


No. 105. STAGE 1.



No. 103; STAGE I.

2 AND 3



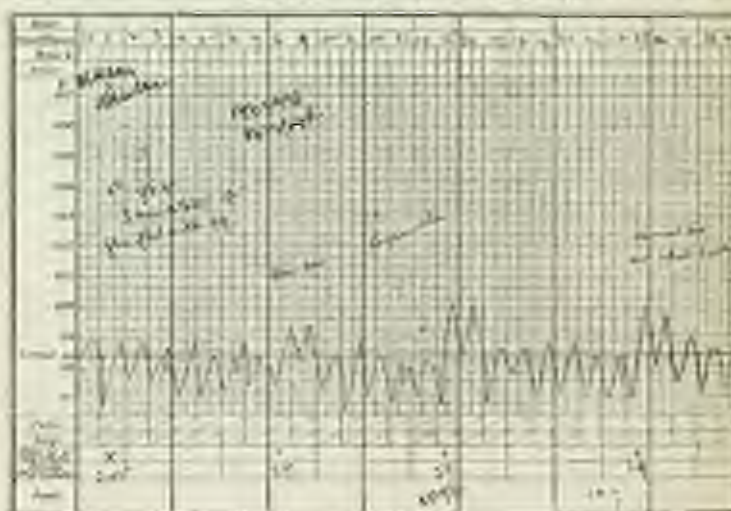
NO. 101. STAGE 1.

4 AND 5

Plot _____ Date _____
 Station _____



Plot _____ Date _____
 Station _____

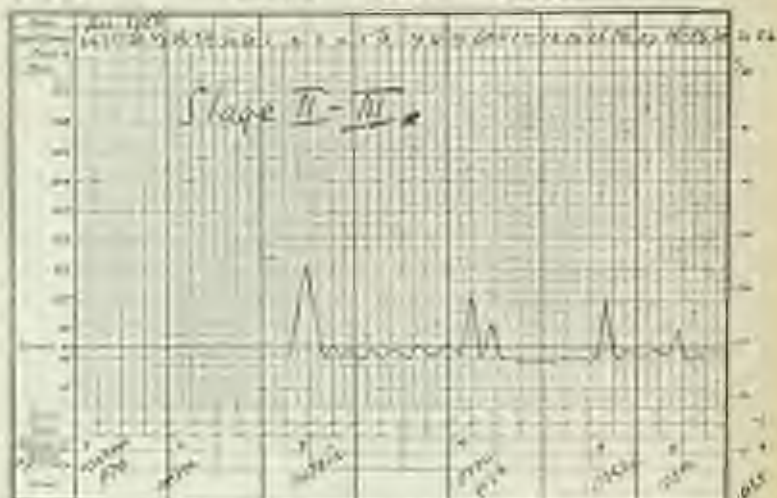


STAGE 11-112.

1 AND 2

Date: _____ To: _____ Amount: _____ Description: _____
 General acct. _____ Received from _____ for _____ \$ _____

Printed on: 11/11/2014 14:14:14



| Name | Age | Weight (lb) | Height (in) | Time |
|-------------|-----|-------------|-------------|------|
| James W. | 18 | 150 | 70 | 1:10 |
| John D. | 19 | 160 | 72 | 1:15 |
| Robert E. | 20 | 170 | 74 | 1:20 |
| William F. | 21 | 180 | 76 | 1:25 |
| Charles G. | 22 | 190 | 78 | 1:30 |
| Thomas H. | 23 | 200 | 80 | 1:35 |
| Richard I. | 24 | 210 | 82 | 1:40 |
| Joseph K. | 25 | 220 | 84 | 1:45 |
| Benjamin L. | 26 | 230 | 86 | 1:50 |
| Samuel M. | 27 | 240 | 88 | 1:55 |
| David N. | 28 | 250 | 90 | 2:00 |
| George O. | 29 | 260 | 92 | 2:05 |
| Patrick P. | 30 | 270 | 94 | 2:10 |
| John Q. | 31 | 280 | 96 | 2:15 |
| Robert R. | 32 | 290 | 98 | 2:20 |
| William S. | 33 | 300 | 100 | 2:25 |
| Charles T. | 34 | 310 | 102 | 2:30 |
| Thomas U. | 35 | 320 | 104 | 2:35 |
| Richard V. | 36 | 330 | 106 | 2:40 |
| Joseph W. | 37 | 340 | 108 | 2:45 |
| Benjamin X. | 38 | 350 | 110 | 2:50 |
| Samuel Y. | 39 | 360 | 112 | 2:55 |
| David Z. | 40 | 370 | 114 | 3:00 |
| George A. | 41 | 380 | 116 | 3:05 |
| Patrick B. | 42 | 390 | 118 | 3:10 |
| John C. | 43 | 400 | 120 | 3:15 |
| Robert D. | 44 | 410 | 122 | 3:20 |
| William E. | 45 | 420 | 124 | 3:25 |
| Charles F. | 46 | 430 | 126 | 3:30 |
| Thomas G. | 47 | 440 | 128 | 3:35 |
| Richard H. | 48 | 450 | 130 | 3:40 |
| Joseph I. | 49 | 460 | 132 | 3:45 |
| Benjamin J. | 50 | 470 | 134 | 3:50 |
| Samuel K. | 51 | 480 | 136 | 3:55 |
| David L. | 52 | 490 | 138 | 4:00 |
| George M. | 53 | 500 | 140 | 4:05 |
| Patrick N. | 54 | 510 | 142 | 4:10 |
| John O. | 55 | 520 | 144 | 4:15 |
| Robert P. | 56 | 530 | 146 | 4:20 |
| William Q. | 57 | 540 | 148 | 4:25 |
| Charles R. | 58 | 550 | 150 | 4:30 |
| Thomas S. | 59 | 560 | 152 | 4:35 |
| Richard T. | 60 | 570 | 154 | 4:40 |
| Joseph U. | 61 | 580 | 156 | 4:45 |
| Benjamin V. | 62 | 590 | 158 | 4:50 |
| Samuel W. | 63 | 600 | 160 | 4:55 |
| David X. | 64 | 610 | 162 | 5:00 |
| George Y. | 65 | 620 | 164 | 5:05 |
| Patrick Z. | 66 | 630 | 166 | 5:10 |
| John A. | 67 | 640 | 168 | 5:15 |
| Robert B. | 68 | 650 | 170 | 5:20 |
| William C. | 69 | 660 | 172 | 5:25 |
| Charles D. | 70 | 670 | 174 | 5:30 |
| Thomas E. | 71 | 680 | 176 | 5:35 |
| Richard F. | 72 | 690 | 178 | 5:40 |
| Joseph G. | 73 | 700 | 180 | 5:45 |
| Benjamin H. | 74 | 710 | 182 | 5:50 |
| Samuel I. | 75 | 720 | 184 | 5:55 |
| David J. | 76 | 730 | 186 | 6:00 |
| George K. | 77 | 740 | 188 | 6:05 |
| Patrick L. | 78 | 750 | 190 | 6:10 |
| John M. | 79 | 760 | 192 | 6:15 |
| Robert N. | 80 | 770 | 194 | 6:20 |
| William O. | 81 | 780 | 196 | 6:25 |
| Charles P. | 82 | 790 | 198 | 6:30 |
| Thomas Q. | 83 | 800 | 200 | 6:35 |
| Richard R. | 84 | 810 | 202 | 6:40 |
| Joseph S. | 85 | 820 | 204 | 6:45 |
| Benjamin T. | 86 | 830 | 206 | 6:50 |
| Samuel U. | 87 | 840 | 208 | 6:55 |
| David V. | 88 | 850 | 210 | 7:00 |
| George W. | 89 | 860 | 212 | 7:05 |
| Patrick X. | 90 | 870 | 214 | 7:10 |
| John Y. | 91 | 880 | 216 | 7:15 |
| Robert Z. | 92 | 890 | 218 | 7:20 |
| William A. | 93 | 900 | 220 | 7:25 |
| Charles B. | 94 | 910 | 222 | 7:30 |
| Thomas C. | 95 | 920 | 224 | 7:35 |
| Richard D. | 96 | 930 | 226 | 7:40 |
| Joseph E. | | | | |

| Journal no. | Journal Title | Year | Vol. | Page No. (s) | Page No. (s) of this |
|-------------|---------------|------|------|--------------|----------------------|
|-------------|---------------|------|------|--------------|----------------------|



STAGE 12-III

3 AND 4



No. 62. STAGE II.

4 AND 2

Min C. This is a typical case—Signs of second stage.
 Temperature in evening 100° to 100.7° for some days. Pulse 110-130. Weight 14 1/2 lbs.
 Treatment started out by increasing dose at intervals varying with reaction—in 3-4 days intervals.
 For every week improvement. Gain in weight and fall in pulse. Such cases prove Wright's theory
 to be probable and method successful.
 See temperatures chart immediately following, with dose given and resulting temperature.

From _____ to _____
 Address _____







Mr. C. 38. Case 30. Stage II. 1906.

Weight 121.2 lbs.

Stage II in printed records (No. 30).

History of 2 or 3 years.

Phlegm—yellow—thick—3j T.B., G. 3.

Short of breath.

Signs. Deficiency of movement, increased vocal fremitus, marked deficiency of resonance over half of upper lobe. Here also bronchial breathing bronchophony. Moist—bubbling and crackling (the upper half of lobe. Ac. apex of lower lobe also involved to some distance.

L. Well-marked deficiency of resonance, increased vocal resonance.

Dec., 1907. Deficiency of resonance, increase of V.F. and R. Bronchial breathing, but no rales. No cough or phlegm.

1907. Gave second course with T.B. increased to full dose (21 cc) from Nov. 13th to Dec. 18th, without a single reaction. Weight, 121.0 lbs.

Feb., 1909. Feels quite well and looks well.

Sept., 1911. Seen pt. in London—very well.

[See temperature chart immediately following, with doses given and resulting temperatures].



III. 1901. Mr. M. 31.

1081. folio.

1. R. II.-III. II.

2.

3. Three years ill.

4. N.

5. V.

6. 90-80.

7.

8. Tb. G. 2: 1901, 100.

9. Bacteriologist.

10.

11.

12. Tuberculin treatment:

T.E. up to 75 cc. Causing many reactions of all grades.

Weight 110. 7 lbs.

"No cough at all for 4 months." Before treatment had

Phlegm, 64-72 instead of 82-90-96. [cough for years.

After one large dose had haematuria and nephritis.

In 1905 reacted. P.T. up to 55 cc.

Pause.

In 1907 reacted again 100. Old T. 100.

P.T. again up to 75 cc. No reaction at all.

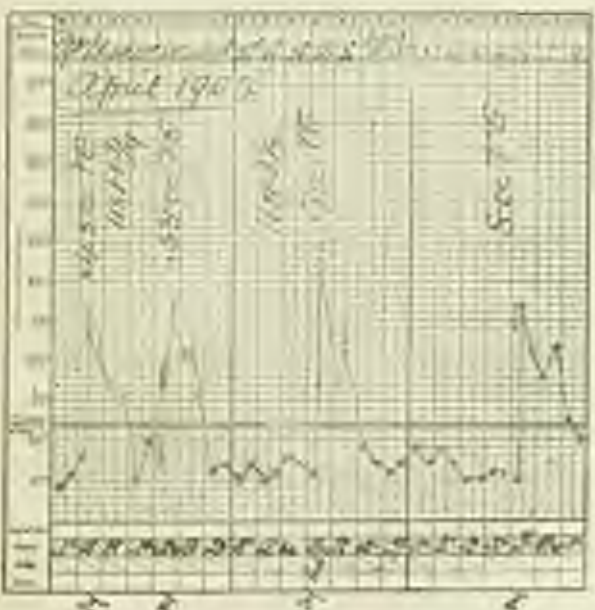
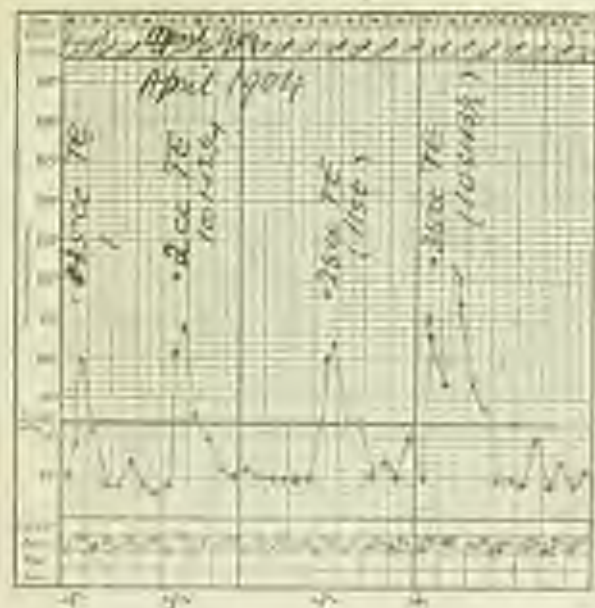
Very great improvement.

March, 1909. Extremely well, feels he can do anything.

[See accompanying temperature chart, with doses and resulting temperatures.]

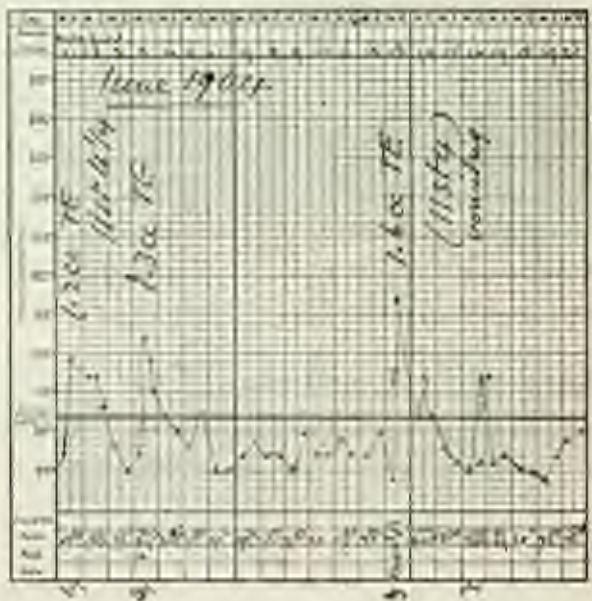
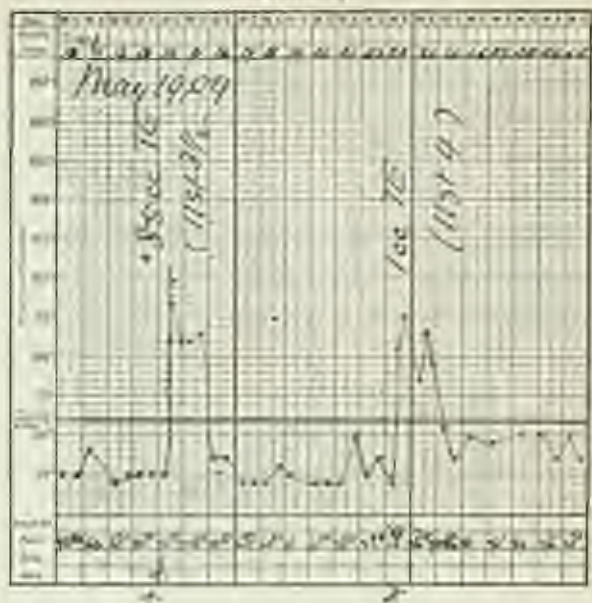
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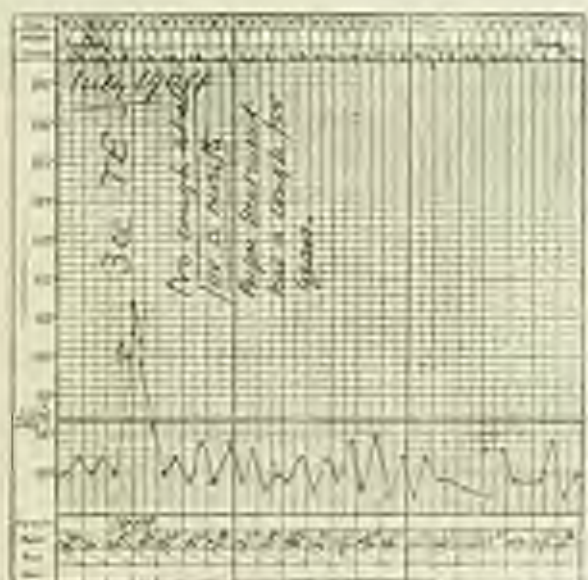




STAGE II-III

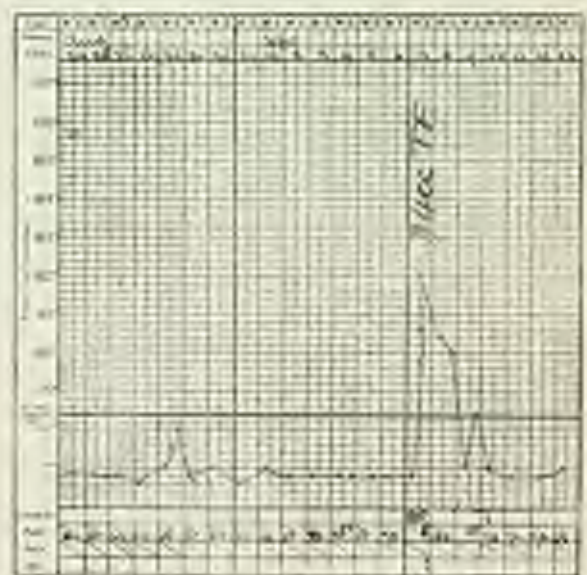
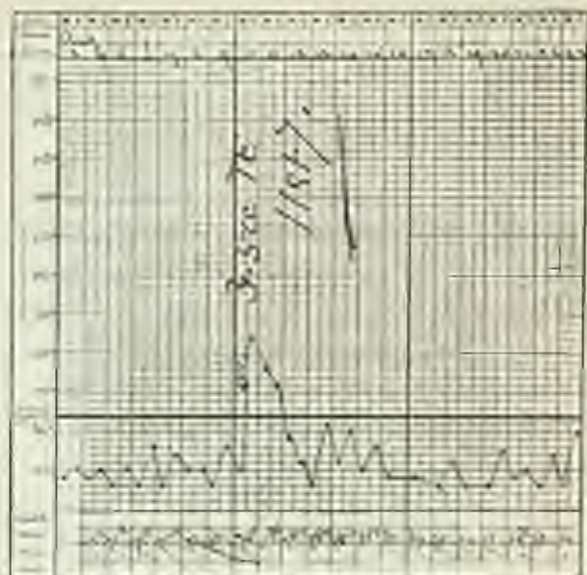
4 AND 5

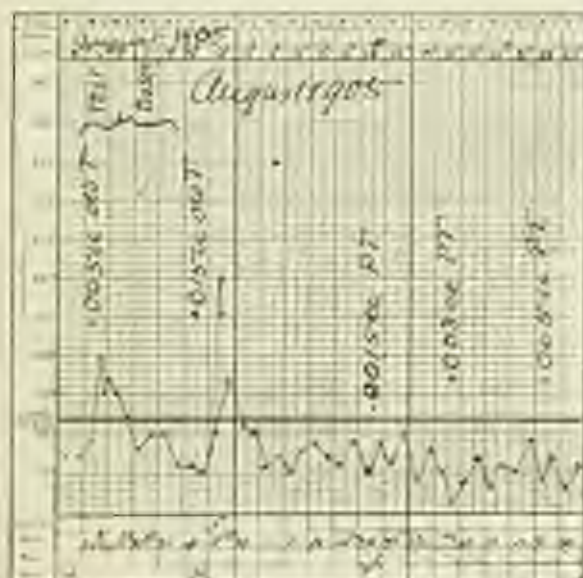




STAGE II-III.

E and Q





STAGE II.-III.

12 AUG 11







Miss G.

See Case No. 13. Stage II-III.

Stage II-III. in printed records.

Her family doctor, Dr. N., admits that the result in this case has made a convert of him.

Tablets, lactu, G. 4-5.

Abundant spores—quite a cupful per diem.

8st. lbs. on coming to me. Now, 2½ years later, 10st. 3lbs.

Before treatment greatest weight 9st. 3lbs.

Good result by increasing doses in spite of reaction against Wright's virus.

Gain of weight from Nov. 4th, 1904, to Feb. 11th, 1906, 8st. 11lbs. to 10st. 0lbs.

Afterwards increased to 10st. 11lbs.

And now (March, 1909) 10st. 6lbs.

Been at work since 1906, and at work March, 1909.

Disregarded Wright's "negative phase" with good result.

Increasing doses in spite of reactions.

Second course:

P.T.O. up to 75 cc. and then P.T. up to 1 cc.

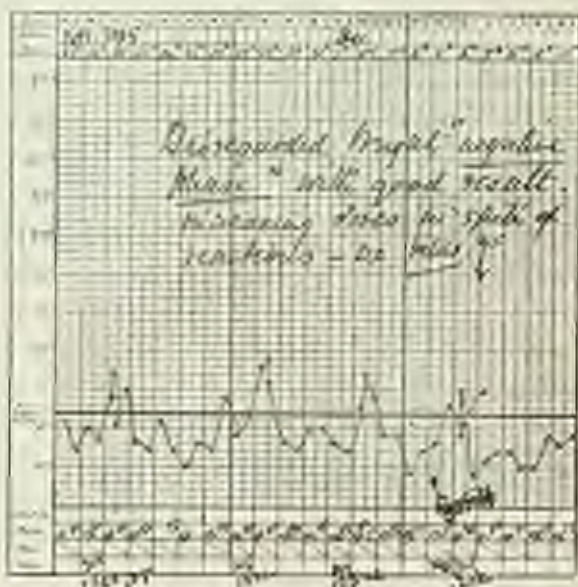
General improvement and not a single reaction.

March, 1909. Weight, 10st. 6lbs.

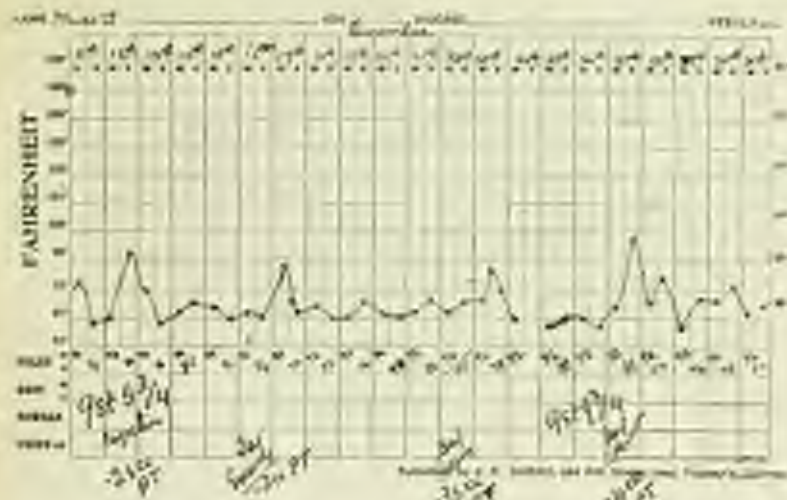
[See temperature immediately following.]

Case No. 13





4 APR 5





No. 100, Strait 1

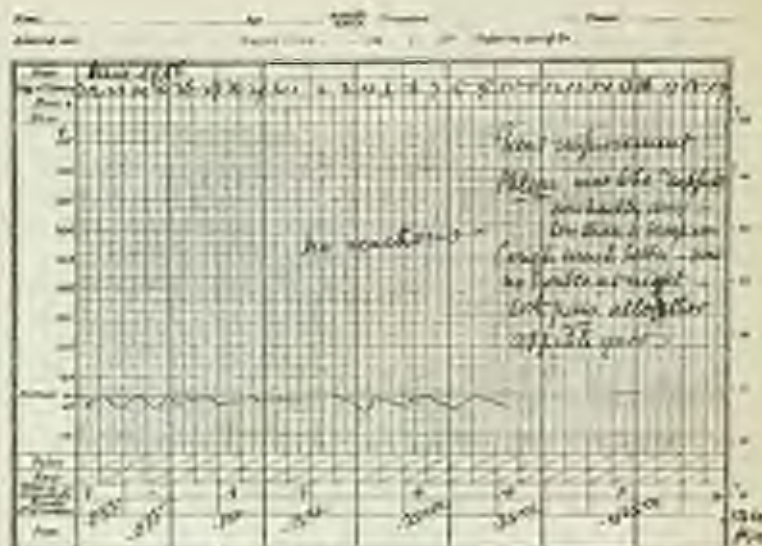
1400 ft.



No. 105 STAGE I.

5. ASD 6





Miss F. 25.
R. I.; L. II.

8th 6th.

A case of extreme over-sensitivity to tuberculin.—The girl persisted in staying at her work in spite of my urgent protests. She was studying Anatomy. Had extensive signs of pleurisy. Temperature, 101°. Also tuberculous arthritis (L. knee) with much pain. She was walking to and from lectures.

After such violent reactions with P.T.O. tried T.R., beginning with 10000 cc.

Break in consequence of going to country. Very great improvement; no pain, no pleurisy. Feeding much better.

March 29th, 1909.

No signs of pleurisy; pain in knee quite gone.

Looks well; appetite good. Weight, 98 lbs.

Still sensitive to T.R., but taking 60 cc.

[See temperature chart immediately following, with doses given and resulting temperatures.]

Treated with P.T.O. in increasing doses (continued) :—

| | | | |
|------|----|----------|----|
| 15 | N. | .005 cc. | N. |
| 18 | N. | .03 cc. | N. |
| 23 | N. | .04 cc. | N. |
| 25 | N. | .05 cc. | N. |
| 30 | N. | .06 cc. | N. |
| 600 | N. | .075 cc. | N. |
| 1000 | N. | 1 cc. | N. |

70c. 2100.

14.5. 06. Glands much smaller, knee free from pain, seems very well.

These are the very cases in which A. E. Wright claims to have obtained such good results by means of small doses of the same amount repeated at long intervals under the control of the optic index, and I have given these details in order to prove to any one who can be convinced by facts that, by increasing the doses at short intervals even when reactions follow the doses, one is able to produce immunity. This record and dozens of other records of mine of a similar nature prove that Wright's practice based upon the optic index and his theory of the "negative phase" is misleading and must lead to failure, unless the mechanism of immunity can be evolved by two radically different methods. By the method of large doses, even at short intervals of a few days, one can reach a degree of immunity which can be gauged either by the great improvement in the symptoms, physical signs, weight, and energy, or, still better, at a subsequent period of nine months to a year after treatment by a negative reaction even to so large a dose of tuberculin as .05 cc.

I doubt very much whether Wright's cases, if tested with tuberculin nine months after treatment, would not react to this large dose of tuberculin, and such a reaction would surely show that by his method he had not reached such a high grade of immunity as by Kott's method. I should be much surprised if cases treated according to Wright's method failed to react even to the dose of tuberculin, which may just cause a reaction in cases immunized by tuberculin (one cc. Old Tuberculin or even .005 cc.). Wright has not used tuberculin to control his results, and if he did, might discover that his theory of the negative phase would not help our practice.

Mrs. R.

70c. 910a.

See Case 16, Stage II.-III. in printed records.

Treated with P.T. up to 7 cc.

Wright's ideas entirely disregarded.

Yet after treatment no cough and no sputum for three months.

After catching a severe cold she reacted again to tuberculin.

A second course with P.T. was given; afterwards weight, 84½/220½.

Tested in October, 1905.

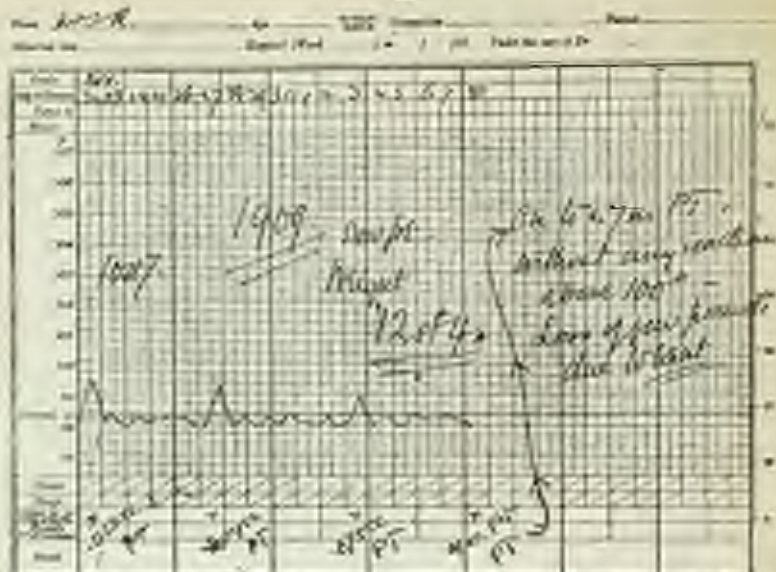
No reaction to .05 Old T.

1909. Weight, 84. 11½. In splendid health.

[Appended is temperature chart with doses and temperatures.]

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Run R



Run R



RESULTS OF TUBERCULIN TREATMENT IN TUBERCULOUS LARYNGITIS

Laryngeal tuberculosis offers the best opportunities for studying the value of tuberculin in the treatment of tuberculosis—at least, in Australia, where, so far as my experience goes, larynx is extremely rare. Two advantages are self-evident. The effects of treatment can be closely and directly watched, and the prognosis of well-marked laryngeal tuberculosis being so utterly bad, any real successes must be attributed to the method. In the successes I have had with tuberculin I have not had recourse to sanatorium methods; and even when the disease of the lungs has reached the third stage I have witnessed ulceration of the larynx completely heal in several cases under tuberculin without any local treatment. In these cases also the lungs have healed in a clinical sense. Cough and expectoration have ceased, and healthy nutrition and bodily vigour have been restored.

Laryngeal tuberculosis is generally a late incident in pulmonary tuberculosis, and secondary to the disease in the lungs. Has any one ever seen a case of laryngeal tuberculosis secondary to a tuberculous lesion elsewhere than in the lungs? Still, laryngeal tuberculosis may be so obviously in advance of the lung trouble that it certainly appears to be primary. I have seen at most three such cases in Sydney, and another recently through the courtesy of Mr. Herbert Tilley.

CASE I.

In the first case there was a circular, raised, ulcerating mass involving the whole of the upper surface of the cord in its middle third. Symptoms of pulmonary trouble were absent. No cough, no expectoration, and physical signs were very doubtful.

CASE II.

The second case was even more remarkable. The epiglottis was severely and extensively ulcerated, and the ulcer extended down to the

base of the epiglottis in its centre so as to form a V-shaped depression. The larynx also was seriously involved. Both cords were hidden from view by a swollen red oedematous mass on each side, especially on the left side, where there was also ulceration of the mucous membrane. The swollen mass so encroached upon the lumen of the larynx that there was stridulous breathing, aphonia, and dyspnoea. The patient, aged sixteen, the daughter of healthy parents, was first taken to Mr. Brady, who told the parents that there was advanced and hopeless laryngeal tuberculosis, and refused to adopt any local treatment. Six months before this the child had been sent up the country, because "she was run down." The child, after Mr. Brady's verdict, was brought to me, and I also at first declined to undertake any treatment. There were no evident signs in the lungs; no cough or phlegm or pain. When I saw the child there was progressive hoarseness, ending in aphonia, and the child looked and felt ill. Temperature normal. Subsequently I consented very reluctantly to take the girl into the hospital, and began treatment with tuberculin at once. I instructed my home-physician to do tracheotomy if there should be urgent dyspnoea. I gave progressively increased doses of P.T.O. and Spengler's E.K. preparation. At first there were reactions to 100 several times, but subsequently large doses were well borne. In two or three months' time the improvement was extraordinary. Both cords came into view and were thickened and reddened, but not dried. The voice returned. The V-shaped fissure filled up with organising tissue; the edge of the epiglottis healed. Meanwhile the girl gained 10 lbs. In another few months the gain in weight was 18 lbs. There was no attempt at open-air treatment, though the child was well fed. The last note (March, 1910) is: "Epiglottis completely healed and edge smooth; larynx apparently normal." Patient feels and looks perfectly well. Next month after treatment patient was tested and did not react to 0.045 c.c.m. Old Tuberculin. Mr. Brady was interested in the case, and I arranged that he should see her again. July, 1911. Girl well and back at school.

CASE III.

The third case showed also definite physical signs of pulmonary tuberculosis in an early stage, and for six months there had been a cough, ending in retching. There was some phlegm, but I could find no tubercle bacilli. Appetite poor. Weight, 82. 12 lbs. Pulse 108. Patient looked very pale and ill. The larynx was seriously affected. There was loss of voice amounting to aphonia. On examination the left cord was more affected than the right, markedly infiltrated and fixed at posterior end, so that it could not be approximated to touch the right cord. There was also superficial ulceration at junction of cord and arytenoid, spreading also into interarytenoid area. The right cord was red and velvety. I treated this case also with P.T.O., and in less than two months her condition was thus described: "The distressing cough

entirely ceased. The voice was fully restored, the night sweats disappeared, and, above all, the weight increased substantially. At the present moment, after a few months of tuberculin treatment, the patient is a different being, to all outward appearance vastly improved in health, with energy renewed in a material degree, and, so far as one may judge from what one sees, on the high road to absolute recovery. This result has been achieved without any resort to the open air." She gained weight. I was able to watch the cords become clean and white, and the ulceration healed.

CASE IV.

The fourth case I saw through the courtesy of Mr. H. Tilley, who sent me the case for tuberculin treatment. The trouble began in the larynx, epiglottitis, and left tonsil. At no time have any obvious pulmonary signs or symptoms presented themselves. The lesion on the epiglottis was accurately excised, and the lesions on the aryepiglottidean fold and left tonsil were treated by galvanopuncture. The lesion in the tonsil was brought to a complete standstill, and subsequently did not react to tuberculin. The result in the larynx was not so complete, as small doses of tuberculin caused marked hoarseness for two or three days.

May 13th. Even now each dose of tuberculin brings on hoarseness. After a time tuberculin failed to produce this local reaction. Sad to say, before the patient came to me for treatment a new focus had appeared on the upper surface of the soft palate just above and to the left of the uvula, looking into the post-nasal space—curiously on the opposite side of the mesial line to the earlier lesions. This focus ulcerated and formed a well-marked excavation with swollen, rounded infiltrated edges and considerable redness and swelling of the surrounding tissues, including the uvula.

At this stage I saw the patient and at once began treatment with tuberculin. No local treatment was adopted, nor any sanatorium measures. After three months' use of tuberculin, which caused decided reactions with every dose, the patient being extremely sensitive to tuberculin, the ulcer slowly healed and the redness and swelling disappeared. I do not presume at this stage to say what the final result may be, because even now the patient is so sensitive that one has not yet been able to give large doses. For a month the patient was treated in London, but since then the patient, having been instructed, is giving herself the injections. The over-sensitiveness persists. I have rarely seen such prolonged over-sensitiveness, and I admit that I have given the doses more cautiously and at longer intervals than

is my custom. I am now asking myself whether the oversensitiveness has been perpetuated because I have increased the doses less rapidly than usual and have repeated them, not every three or four days, but every seven or eight days. In order to see whether this is so, I am now giving a dose which I hope will cause little reaction, and I shall repeat this dose every two days.¹ The patient writes that, in spite of the reactions, her throat feels quite well and she feels well in herself. Still, after the doses there is temporary hoarseness. I shall not be satisfied till I have given large doses without any reaction, and then after an interval of nine months have tested her. Whether I shall succeed with tuberculin where the most skilful surgical measures have failed remains to be seen. While she was under Mr. Tilley the patient had open-air treatment at St. Moritz. It will be extremely interesting to watch in this case the effect of tuberculin alone. The patient cannot afford to remain under constant observation.²

So much for the tuberculin treatment of tuberculous lesion occurring early in the larynx.

Laryngeal tuberculosis, secondary in incidence and appearance to pulmonary tuberculosis, is a capricious manifestation occurring in any stage of lung disease, and with increasing frequency and intensity in the later stages.

In 150 cases of pulmonary tuberculosis in the first stage I have seen but a few cases in which the lesion could be definitely described as tuberculous. Laryngeal catarrh is a common incident in early pulmonary tuberculosis, but unless either the appearances in the larynx are distinctive or the laryngeal lesion reacts to tuberculin I do not describe them as tuberculous. There is a pre-tuberculous laryngitis of a simple kind. After treatment with tuberculin I have never seen a tuberculous development in the larynx in these early cases. Case II, above reported, belongs to this category.

In another instance an accomplished singer consulted me for "cracked" voice. I found absolute and hopeless paralysis of the right

¹ See now following this article, p. 394.

² November 2003: Patient has taken 1 c.cm. of P.T., weight 100, lbs., voice normal, reactions to tuberculin have entirely ceased.

vocal cord, due to injury of the recurrent laryngeal nerve by a tuberculous lesion of the pleura and apex of the right lung. This symptom led me to examine the apex of the lung, where I found signs of early tuberculosis. The first test dose of 0.001 c.cm. Old Tuberculin raised the temperature to 101.6° and settled the diagnosis. Treatment with tuberculin healed the lung, but the cord was permanently paralysed.

The third patient was also a fine singer, who completely lost her voice with the onset of the disease. The changes were not very distinctive, but she reacted energetically to 0.001 c.cm. Old Tuberculin, and was then treated with T.E. up to 4 c.cm. There were obvious signs at the right apex. She lost all her symptoms, gained a stone, and regained her voice. Some years after I heard her sing at the Sydney Town Hall. She married later, and was quite well in 1909, six years after I treated her.

CASE IV. long suffered from hoarseness. Reacted to tuberculin. Treated with P.T. up to 0.775 c.cm., working all the time. Completely recovered health and voice. A year after treatment did not react to 0.045 c.cm. Old Tuberculin. Well one year later.

Accordingly the typical lesions of laryngeal tuberculosis are rare in the early stages of pulmonary tuberculosis, and in my experience invariably yield to tuberculin treatment alone.

In the second and third stages of pulmonary tuberculosis when tubercle bacilli are being constantly discharged by the air passages, typical lesions become more and more frequent. Statistics show that the larynx is affected in nearly 50 per cent. of the late stages of pulmonary tuberculosis. Sanatorium statistics must be ignored, because laryngeal tuberculosis disqualifies for sanatoriums. In German sanatoriums (Hamel), out of a total of 7258 men and 2413 women, there were five cases of laryngeal tuberculosis among the men, and none at all in the women. Sanatoriums have to live and keep up their reputation by records, and are forced to protect themselves from utter failure by rejecting unfavourable cases. The excuse given for rejecting laryngeal cases is that high altitudes are bad for these cases. It would be nearer the truth to say that laryngeal cases are bad for sanatorium statistics.

In ninety cases of pulmonary tuberculosis in the second stage, I have had fifteen very definite laryngeal tuberculosis, six manifesting severe and persistent hoarseness with red-

ness, swelling, and thickening of the cords, while nine others had both infiltration and ulceration. Of course when severe laryngeal tuberculosis complicates the second stage of lung disease, the case should be classed with those of a later stage.

Yet in all the six cases of the milder form there has been complete and permanent recovery of the voice, with return of the cords to a healthy state. In the cases with ulceration I have watched the ulcer slowly heal.

In one severe case a circular ulcer nearly as large as a threepenny-piece developed between the arytenoids. I watched the ulcer slowly heal till not a trace of it was left. This occurred *only* in my experience when I was still using lactic acid (50 per cent.). The lactic acid seemed to do positive harm—so I gave it up, and have not used it since. The lung also, though in a late second stage, improved. Cough and expectoration ceased and tubercle bacilli disappeared from the sputum.

For four years the larynx remained well, but the patient was very poor, and tried to make a living by teaching deaf-mutes lip-reading. Her health broke down again, and she died of pulmonary tuberculosis eight years after she had shown a severe lesion of the larynx in the second stage or worse of pulmonary tuberculosis.

In all the other cases the improvement was extraordinary. One of them had been treated by Mr. Percy Kid in 1905. I saw him in March, 1906.

Then the lungs were R. II., L. II.-III. Sputum 0; Tn. G. 4. There was much infiltration of cords, especially left. I gave him several courses of tuberculin. The improvement in the lungs, in general health and in larynx was very great. Unfortunately a band of fibrous tissue formed across the anterior angle of the larynx, which seriously injured the voice. The patient was a salesman and had to do much talking to get a living. Otherwise he was reported as well at the end of 1909.

In another case an ulcer formed on false cords with much swelling, which healed, but there was also ulceration of cord and aryepiglottidean fold, which healed and left a firm band of organised tissue which fixed the right cord and seriously affected the voice. In this case the result was not satisfactory, as the disease in the lungs progressed, and in two years the patient died.

Case IV. illustrates well the common history of cases treated at sanatorium.

July, 1903, sent to sanatorium with note: "No physical signs and no tubercle bacilli." "Had previously lost 30 lb. in a few weeks." At sanatorium gained 25 lbs.: "one of their best cases." Discharged and went to sea. In five weeks lost 25 lbs. Came to me. State of lungs, R. L. Tubercle bacilli, G. 5. Patient looking ill and very weak. Treated with tuberculin while living in poor part of Sydney. No sanatorium treatment; improved immensely. Lost cough, recovered appetite and strength. No relapse while under my observation (more than a year). Came to see me every few months. Larynx gradually returned to normal condition. The cords had been red and inflamed and were rough and gritty.

CASE V.

Case No. 49, p. 209, in records published in book. Lungs stage, R. II.; L. II. Temperature 100° to 100°. Hoarseness, diarrhoea, and griping pain for nearly three months. Later double ischio-rectal abscess. Treated with tuberculin P.T.O. to 1 c.cm., then P.T. to 0.15 c.cm., and later up to 0.7 c.cm., 25 lbs., and recovered voice. Very well a year later.

CASE VI.

Lungs, R. II.; L. II. Appetite poor, looks very ill. November, 1907, very hoarse. Lungs, R. II.; L. II. Th. G. 3 in large clumps. Treated with P.T.O. and P.T. Many reactions, 100°, 100½°, 101¼°, 100½°, 101°, yet good improvement. Gained more than 9 lbs. (lost 5 lbs. to 30 lb. ill). Voice restored; cords healthy. March, 1910, very well.

CASE VII.

State of lungs, R. II.; L. L-II. Sputum, 3 or 4 Th. G. 9. Night sweats. Temperature 100½°-103½°. Country air two years. Disease began while in country. Came to Sydney. Sent to mountains, got worse, and plainly supervened. Marked tuberculous laryngitis with redness and infiltration. Treated with tuberculin. March, 1910. Very well.

CASE VIII.

Whole or upper surface of epiglottis converted into a thick, granular, elevating mass; there was marked infiltration of cords and a small inflammatory ulcer causing complete aphonia. Lungs, R. II.; L. L-II. Sputum, 200; Th. G. 5. Tuberculin P.T.O. to 1 c.cm. Then P.T. to 0.5 c.cm. Epiglottis healed completely and cords recovered. Voice returned. No cough or phlegm. Gained 25 lbs. Tested six months after, no reaction to 0.05 c.cm. Old Tuberculin.

This man lived five hundred miles from Sydney, and gave himself most of the doses.

CASE IX.

State of lungs, Stage II, Th. G. 4. Hoarseness due to tuberculous laryngitis, chiefly infiltration of cords and interarytenoid area. Treated with P.T.O. and P.T. Many reactions, 102°, 102°, 104°. Yet six months after "feels very well, physical signs almost disappeared, nothing else abnormal."

This case has not been long enough under observation to justify any opinion as to the ultimate result.

But gratifying as these results have been, they are not to be compared with the extraordinary results I have seen in pulmonary tuberculosis later than Stage II, in Stage II-III, and Stage III, obtained by using large doses of tuberculin with no sanatorium methods at all, without any local treatment, and with a total disregard of the opsonic index and the curious negative phase.

CASE I.

Aged 32, weight, 301 lbs. Came to me in December, 1902. Lungs, R. III (cavity); L. I-II-II. Treated by thirteen doctors. Abundant sputum, many osicles. Th. G. 6. Very hoarse. Obvious infiltration of cords, especially left, involving interarytenoid space. Later definite ulceration of cord occurred which spread to interarytenoid space. Gave several courses of Old Tuberculin, T.E., P.T.O., T.O.A., P.T., and Old T. Ulcer completely healed—all signs of active disease in lungs disappeared. Patient gained over 40 lbs., and has been working in a newspaper office for several years. I saw him in 1909, seven years after treatment, in robust health, and with a perfectly normal voice. His sister, affected after him, had sanatorium treatment for ten months—much improved, but dead in less than two years from onset of illness. She had no tuberculin treatment.

CASE II.

1904. Boy, aged 17, weight, 711. 10 lbs. Had been under Dr. S. S., in Sydney, for two years. "Took pounds of Dr. S.'s medicine, cod-liver oil, and maltine." Always lived in open air. For six months travelled forty miles, then forty miles back once every week to the doctor. "But cure" with a vengeance, then sent inland. "Got much worse." Then his father brought him to me. State of lungs, R. III.; L. II. Numerous Th. and mixed infection (streptococci). Pulse 110 to 120. Temperature, 100° to 101°. Abundant sputum. Very obvious laryngeal tuberculosis, causing great hoarseness, cords reddened, ridged and ragged. At urgent request of father and boy treated with tuberculin, P.T.O., and then T.E. up to $\frac{3}{4}$ gramm. Many reactions, 100°, 100.6, 101°, yet gained weight, 10 lbs. The boy said, "Not two coughs a day since last dose, not a bit

of phlegm. Oh, it was grand." He gained another ribba, and pulse was 80.

In 1906 I tested him up to 0.05 c.cm. (Kd Tuberculin, and he did not react. The larynx was healthy and voice recovered. Two years later he was well.

CASE III.

1904. Patient had had open-air treatment at Orange for one year and a quarter under several doctors. Came to me an absolute wreck early in 1904. He could not speak, great breathlessness, and cyanosed. Temperature 100.5°, 101°, 102°. State of lungs, R. III.; L. II. Abundant thick yellow sputum. Th. G. 5. His doctor left for England and visited patient's mother to inform her that her son could not live more than a month or two. He also diagnosed "necrosis of the vocal cords"—quite rightly. Dr. S. S. ordered cod-liver oil and a quart. At first I refused to treat him, but he implored me to try, and I yielded.

Treated with P.T.O., and then three courses of P.T. up to 0.75 c.cm. He gained 3½ lbs. He was about eighteen months under treatment.

1908. Saw him in excellent health and spirits. No cough, no sputum, and a good robust voice.

1910. Managing a rubber plantation in the Malay States. "Never was better in his life."

I have only to point out also that tuberculin had this marvellous effect in spite of regular fever, as in the previous case, in order to show the profession that even in hopeless cases wonders can be performed. If one can do this in hopeless cases, is it any wonder that one always succeeds in easy and mild cases?

CASE IV.

Had been treated for two years without benefit. Came to me. State of lungs, R. II-III.; L. I-II. Sputum, 400 daily; Th. G. 6; pulse 96. Infiltration of one vocal cord; later atelectasis. Complete loss of voice.

Treated with T.R. In spite of great sensitiveness and severe reactions (103°, 102.6°, 103°, 105°, 105°), result was "no cough or expectoration, very good appetite, and complete recovery of lost energy." This result obtained without local treatment to larynx, and without any open-air methods. He lived near Sydney, and, like all the other cases, was treated as an ambulatory case. Four years later he looked and felt well, but as he reacted I gave him P.T. up to 0.85 c.cm.

CASE V.

Young man, aged 22. State of lungs, R. II-III.; L. II.; sputum 400; Th. G. 8 in numerous clumps; mixed infection streptococci; larynx—advanced tuberculosis, both cords red and ragged, with swelling over arytenoids. Three months before he came to me Dr. S. S. of Sydney

said it was an early stage, and "there was nothing to worry about," and sent him up the country. Certainly he gained flesh, but meanwhile the disease advanced almost to the third stage, and for the last two months he had completely lost his voice.

I treated him with T.R. and T.E. He recovered his voice, the larynx healed, and the sputum was a dessert-spoonful instead of half a cupful. Unfortunately he gave way to drink, and two or three years later I heard he had had a relapse. He was alive for several years after I treated him.

CASE VI.

Young girl, aged 17, came to my ear in 1900. State of lungs, R. III.; L. I. to II. Pulse 120 to 120. Temperature 100°. Abundant expectoration, Th. G. 5. Tuberculous laryngitis of mild form. Treated with T.E., P.T.O., P.T.

1904. Patient looks very well, has recovered all old energy, can sing well, and has no cough or expectoration. Pulse normal.

1910. Eight years after she was told she was dying, she has had a slight relapse, but is responding well again to tuberculin treatment.

For many years I have given up all other methods of treating laryngeal tuberculosis except by means of tuberculin. No drugs—though Sir Felix Semon seems to approve of creosote. No open-air treatment, no local applications except palliatives for pain, no surgical measures such as lactic acid, excision (Heryng), galvano-puncture—and my results have been in some cases quite extraordinary. In cases in the first stage, although some date back for twelve years, I have not seen laryngitis supervene in a single case. Certainly in two cases in the second stage there was a relapse after six years' complete freedom from symptoms, and in both these cases laryngitis soon developed and death followed. In both these cases, in spite of my advice that they should be tested from time to time, they put off coming for six years, and then I could do nothing for them. In most of the cases in which tuberculous laryngitis existed, whether in II., II.-III., or III. stage, great improvement followed, and for several years there has been no return, while in the third stage I have seen even ulceration of the larynx heal. The series of cases in which I have carefully watched for many years the immediate and after-effects of tuberculin treatment according

to clinical and not laboratory methods, leaves no doubt in my mind that in tuberculin we have a remedy not only better than all other known methods combined, but far the best that is likely to be suggested for use at this or any future time in all cases of laryngeal tuberculosis, even in the most hopeless of cases. Nothing has done so much to strengthen my firm conviction of the value of large doses of tuberculin in pulmonary tuberculosis as the extraordinary effects I have closely watched week after week, and year after year, in laryngeal tuberculosis.

Appended is the chart showing temperatures, doses and weight during treatment of Case IV., p. 386.

The opportunity of treating this case of primary laryngeal tuberculosis with tuberculin was given me by Mr. Herbert Tilley. The patient had been under him for some time. The trouble began, as far as I remember Mr. Tilley's account of it, in the aryepiglottidean fold. There was also a deposit in the epiglottis which was excised, and an ulcer in the left tonsil. Excision of the focus in the epiglottis and galvano-puncture of the lesion in the left tonsil and left aryepiglottidean fold, brought the disease to a standstill for a time; but within three months a further deposit occurred on the upper surface of the soft palate which defied surgical treatment. Then it was that Mr. Tilley sent this patient to me.

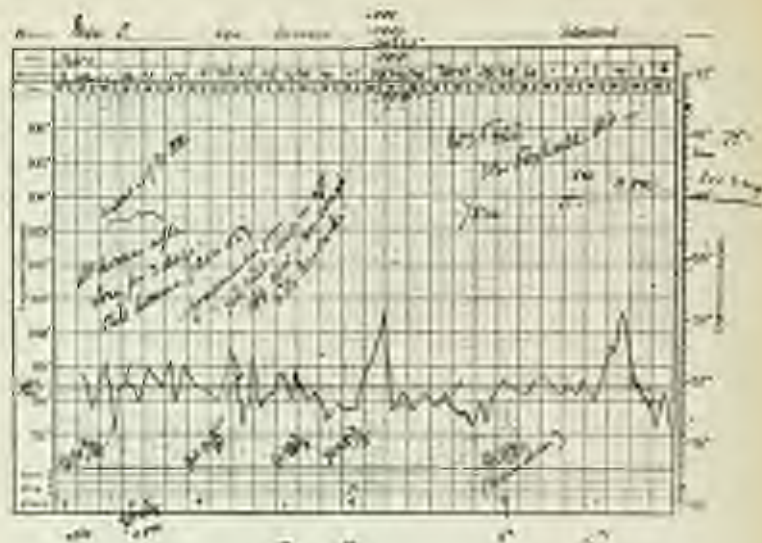
The first dose of tuberculin caused increased hoarseness and pain in the larynx, which slowly abated. Evidently, there was still tuberculous trouble in the larynx, but within two months of using tuberculin the ulcer on the upper surface of the palate was almost healed. Shortly after the beginning of the treatment two granulomata developed in the anterior fissure—one about as large as a split pea, and the other more elongated and hardly measuring more than a fifth of a cubic centimetre in diameter. I sent the patient then to Mr. Tilley, and expressed the wish that he would not be unduly anxious. I continued treatment, and gradually these

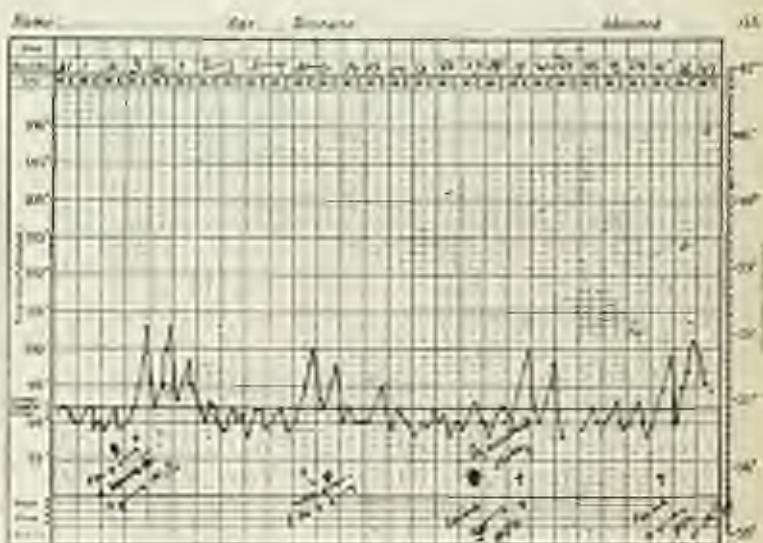
granulomata literally melted away and disappeared. By this time the ulcer in the palate was completely healed, and the palate regained its normal appearance and condition.

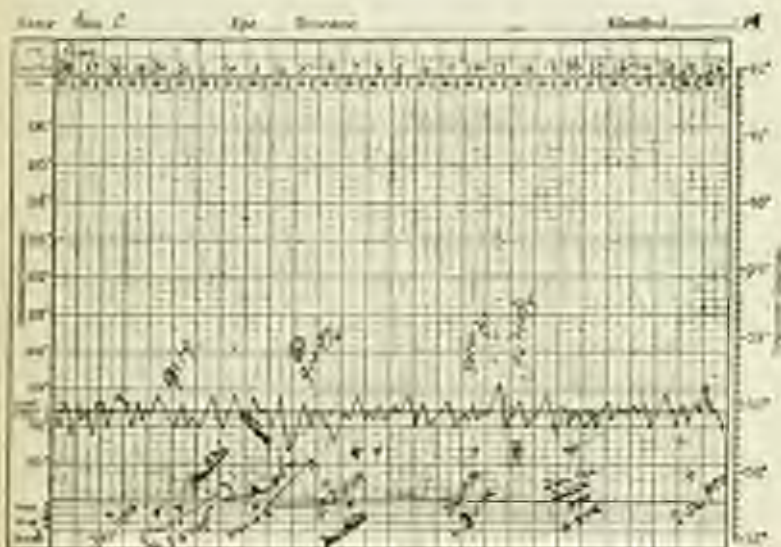
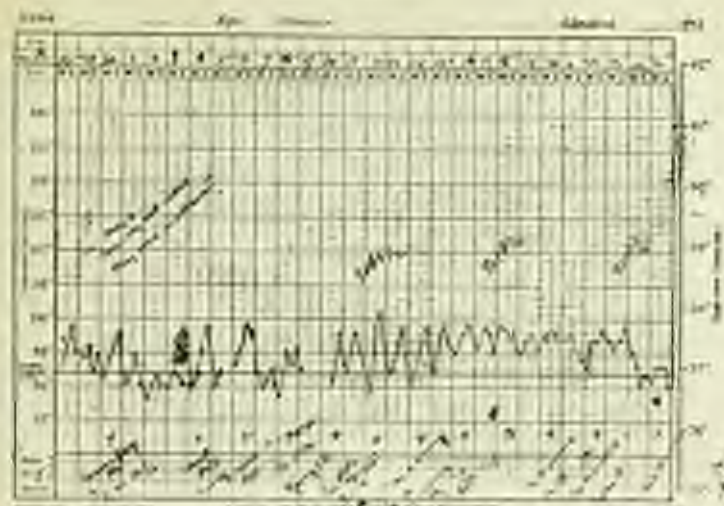
For two or three months (see chart) this patient exhibited great sensitiveness to the doses. I repeated a small dose of about $\frac{1}{8}$ cc. of P.T.O. about once a week, but it always caused a temperature of 101° or thereabouts. During this time the patient was herself injecting the doses which I sent regularly. With this extreme over-sensitiveness persisting, I was forced to ask the patient to come up to London, and then I reduced the dose a little, and repeated it every second day for about three weeks. Thereafter, the course of treatment went smoothly enough. I was able to increase the P.T.O. up to a cubic centimetre, and then give P.T., and finally Old Tuberculin up to one cubic centimetre without any disturbance of temperature worth speaking of.

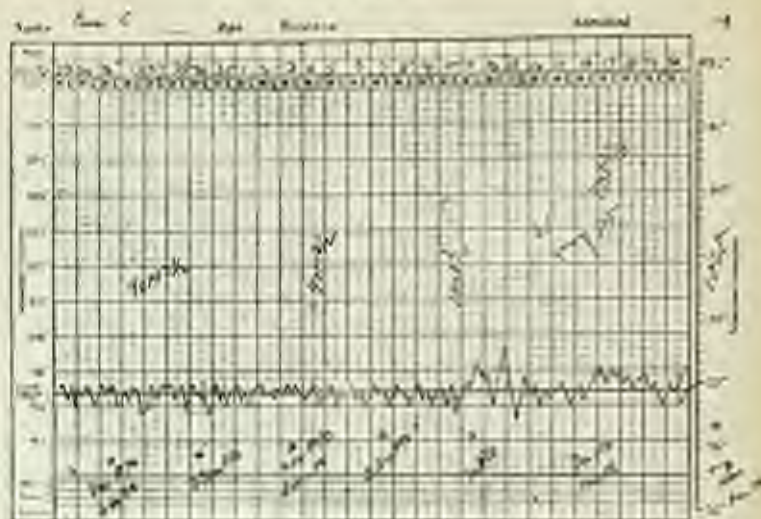
Perhaps nothing shows better the manner in which over-sensitiveness (anaphylaxis) may be encouraged by giving doses even at intervals of a week, and, on the other hand, may be entirely obviated if a similar dose is repeated at intervals of two or three days. It is quite impossible to reconcile this concrete instance of anaphylaxis with Wright's theory of the negative phase. I had long ago observed that by giving too small doses and prolonging the interval between the doses this phenomenon of anaphylaxis was favoured rather than thwarted. The ultimate result in this case was that at the end of about eight months' treatment every vestige of tuberculous disease had disappeared.

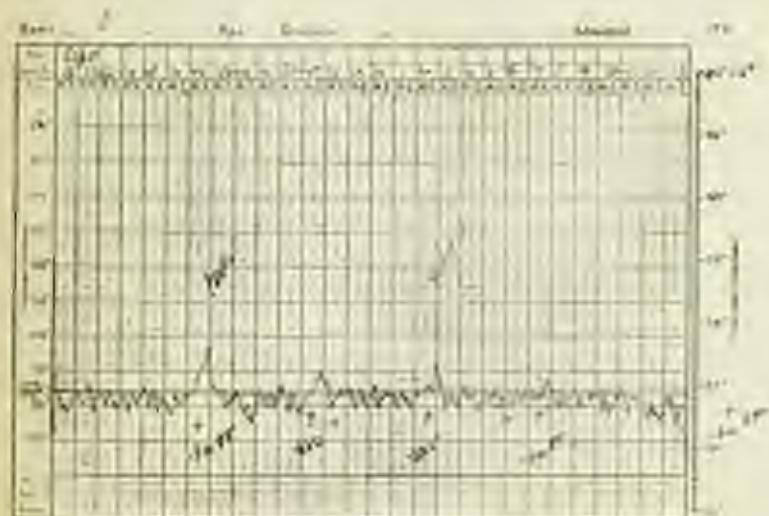
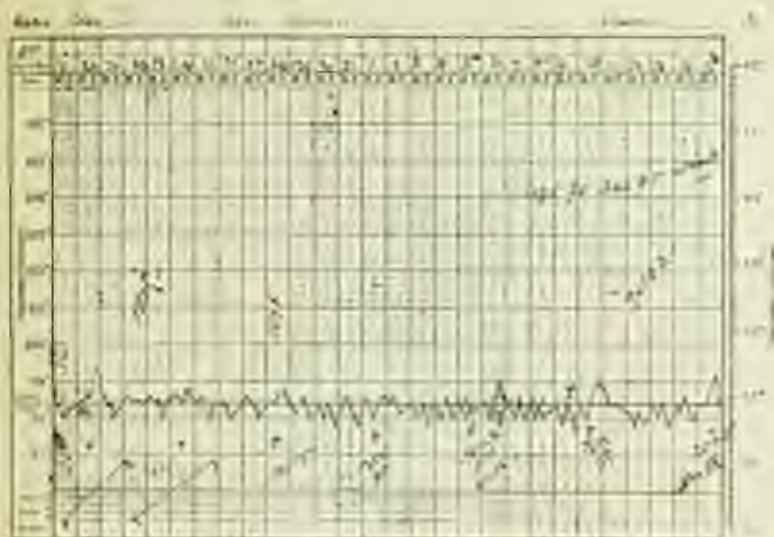
Until this patient was placed under tuberculin treatment she had had the advantage of the highest skill in surgery, and the best open-air treatment at St. Moritz. These measures had hopelessly failed, and even when the condition seemed to be beyond the reach of any remedy, by the use of tuberculin alone, without any surgical treatment, and without sanatorium methods, the patient was restored to health.

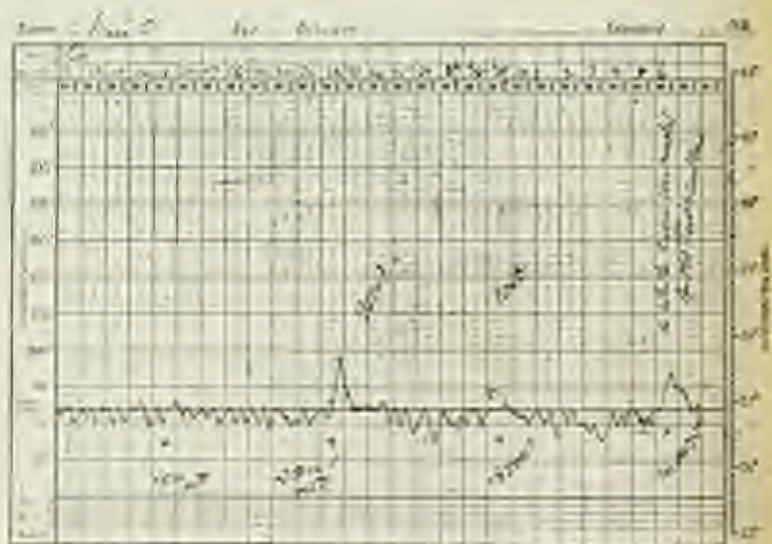
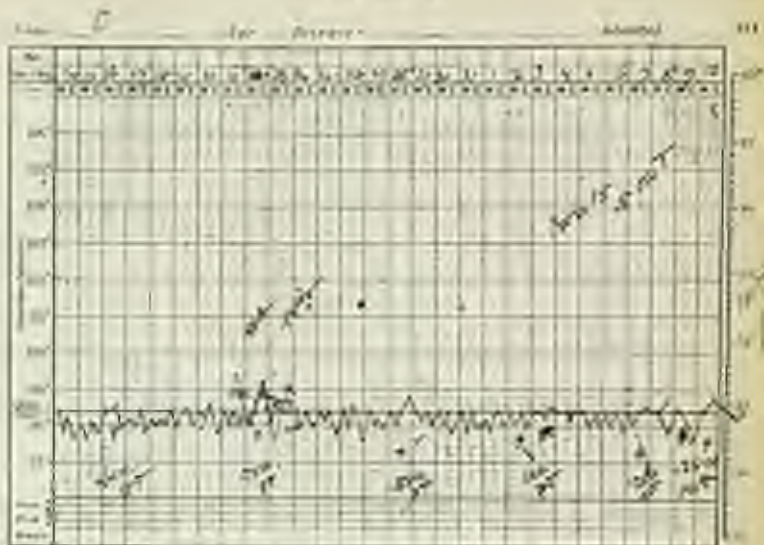














RECORD OF POST-MORTEM EXAMINATION OF A CASE IN THE THIRD STAGE, TREATED WITH LARGE DOSES OF TUBERCULIN.

Case 6. Stage III. In printed records. Brief statement of treatment (see Chart, page 405, etc.).

1904-5. Treated with P.T. In spite of reactions gained 12 lbs.

1906. Reacted. Treated again with P.T.O. (1 cc.) and P.T. up to 65 cc.

1907. Several attacks of sub-acute pleurisy. Reacted. Treated with T.E. up to 8 cc.

1908. Treated again with P.T. up to 95 cc., no reaction above 100°. Very great improvement in physical signs. R. lung: upper lobe mostly solid, with cavity. L. lung: upper lobe almost solid, no rales, except occasional ones over cavity. Sputum, only about 2 drachms. Temperature and pulse normal. Of course, always short of breath, but looks and feels relatively well.

1909. January. Sudden accident. Air escaped into mediastinal tissues and, pressing on heart, caused death (see post-mortem notes).

Patient complained to nurse of pain over cardiac region just to the left, followed by dyspnoea, and slowly increasing embarrassment of heart, ending in slowly progressing general dropsy. Pulse rapid and soft. No albuminuria. Temperature, 99° to 99.4°. I saw patient ten days after onset (on my return from Tasmania). I concluded at once that something was interfering with the action of the heart as a suction pump. By physical signs I excluded effusion into the pericardium, and on percussing I found a resonant note in front of heart. I then thought it possible that there were extensive pericardial adhesions, especially as

there had been so much extensive organising pleurisy on the left side.

The patient died the next night, and I felt sure that something had happened which prevented the proper action of the heart.

Post-mortem Conditions.

With exception of lungs, organs (brain, liver, spleen, kidneys) seemed fairly healthy and well nourished, except that there was intense passive hyperæmia. The heart was enlarged. The right auricle (especially auricular appendix) and ventricle were greatly distended with coagulated blood, while the left side, though dilated, was empty. In front of the heart there was *extensive emphysema of the anterior mediastinal tissues*. This was very marked, and at once attracted notice. In the left lung there were several dilated emphysematous bullæ, larger than a walnut, and one of these projected inwards towards the heart for quite an inch.

There were also many so-called lung "infarcts," of squarish form, purplish black solid areas, showing, on microscopic examination, tissues and bronchioles full of blood. Nothing could show more clearly that these masses were not infarcts at all—the old idea till Prof. Hamilton, of Aberdeen and Grawitz, showed them to be caused by back pressure from obstruction in the left side of the heart. Such "infarcts" occur in mitral disease, especially mitral obstruction. In this case there are these two unusual conditions—*emphysematous bullæ of large size* and *emphysema* in the tissues around the heart on the one hand, and these "*infarcts*" on the other. What is their relation to each other? They could not be due to any third cause—for what third cause could produce them? They must therefore be related as cause and effect. Infarcts could not cause such localised emphysema. On the other hand, emphysematous bullæ rupturing near the heart would allow air to escape and slowly produce an elastic air-cushion, which—under the special circumstances when extensive pleuritic adhesions would prevent a general pneumothorax—might certainly and, in my opinion, did actually interfere with the action of the left side of the heart as a suction pump. This view of the conditions—not far

fetched, when one remembers the extensive emphysema of tissues, can alone account for the failure of the left side of the heart, with consequent damming back of the blood, numerous "infarcts," engorgement of the right side of the heart, all cavities (right ventricle, auricle, and auricular appendix) being enormously distended, general venous congestion with engorged liver, kidneys, etc., and slowly progressing oedema of limbs, of face, and of whole body. In fact we have reproduced in this case the very mechanical conditions that occur in cases of pericarditis with effusion (see Cohnheim's lectures on general pathology) except that the right side of the heart was also engorged. The lungs were also engorged with blood. Accordingly the hindrance to the circulation must have been on the left side of the heart; and in the absence of mitral disease (see specimens showing mitral valves efficient) the only explanation of such conditions seems to be interference with the action of the left side of the heart by the elastic cushion of air formed outside of the left side of the heart by the escape of air from the lungs into the mediastinal tissues—in fact, a localised pneumothorax which, in any other situation, would not necessarily have caused death. Perhaps even the pulmonary veins were pressed upon.

But apart from these interesting anatomical conditions our chief interest is centred in the effects produced in the diseased lungs by large doses of tuberculin. Throughout both lungs, but chiefly in both upper lobes, there is extreme and extensive fibroid organisation of the tissues, from young-celled tissue containing small and large organising elements of every shape, to dense fibrous areas, both peribronchial and interstitial in distribution. Here and there large multinucleated cells are seen which show no sign of degeneration. Nowhere have I found by the naked eye or microscope the least evidence of caseation. In the right upper lobe there are scattered areas of small-celled infiltration visible through the microscope. Even by selecting the most promising portions and staining thin sections stained by Ziehl's method I could not discover a single tubercle bacillus in the tissues. One isolated colony of tubercle bacilli enclosed mostly in

cells was found only in the practically necrosed layer bounding a cavity in the apex of the right lung. This cavity is about the size of a small orange, at least two inches in diameter, and is bounded on every side by a firm, thick, well-defined dense fibrous layer, completely shutting off the cavity from the functioning lung tissue. Nowhere else could I find a typical tubercle or evidence of caseation. Even on opening up the septa no trace of a tubercle or caseation was visible. Nor was there any sign of a tubercle in the brain or kidneys or other part. An examination of the lungs, which I send herewith for examination, shows the enormous extent and degree of fibrous organisation, especially in both upper lobes. Surely one may say that this fibrous organisation is the best anatomical expression of the great effort made by the tissues to resist the action of the tubercle bacilli, and when one contrasts the conditions present in this case with the conditions usually present in advanced stages of pulmonary tuberculosis, it is difficult to avoid the conclusion that the difference is mainly due to the large doses of tuberculin that were used in the treatment of the case.

It is perfectly clear from an examination of the lungs that this patient did not die of pulmonary tuberculosis at all, but rather from a complication that had its origin in the destruction and weakening of tissue (emphysema) left by the disease, after the disease had been arrested. I have already pointed out that even severe hæmorrhages (Case 47, Stage II.) may occur in the lung and air passages when the disease has come to a standstill. This patient had terrific hæmorrhages, without any other evidence of tuberculous disease (no phlegm, no temperature), and yet ever since, after a lapse of more than 2½ years, he has remained perfectly well. No kind of treatment can prevent such accidents. This post-mortem examination offers to us the most powerful vindication of the enormous virtue of tuberculin in large doses—given more or less according to the system which Prof. Koch himself has advocated. Moreover, this experience and the series of cases in Stage II., Stage II-III., and Stage III., treated with large doses of tuberculin in spite of severe reactions (see series of cases immediately preceding), surely proves that mobilisation

of tubercle bacilli is not a danger to deter us from practising this rational method of dosage which secures such striking results. In grateful remembrance of this patient, who was a real hero in life and in death, I can but add that he was one of the finest types of man it has been my privilege to know. He died beloved of all who knew him, especially little children, who were fascinated by his charming personality. His death is to some purpose if this history leads others to have the same firm and unshaken belief in the virtues of tuberculin which possessed him to the very end. He was a man of the finest intelligence, and if many painful blows have been aimed at me because of my staunch advocacy of tuberculin treatment, the friendship and confidence of our such man goes far to compensate me for much suffering. He wished to live, but was not afraid to die. More than anything else in my long experience the accidental death of this attractive man has taught me what a boon we possess in tuberculin, if we use it with courage and intelligence.

Herewith are sent post-mortem specimens:—

1. Upper lobe of right lung showing cavity.
2. Left lung—upper lobe showing extreme fibroid organization of old tuberculous areas, also extensive pleuritic adhesions and numerous "infarcts."
3. Heart—enlarged as is usual in such conditions of lung and pleurisy. Muscular tissue and valves healthy.
4. Kidneys healthy but for passive congestion.

Finally numbered *microscopical sections* of wall of cavity and various parts of the lung showing:—

1. Fibroid changes in all stages, emphysematous areas, and everywhere engorgement of the tissues with blood, including the numerous "infarcts" with blood in tissues and air-passages.
2. Tubercle bacilli stained by Ziehl's solution only in one focus in the necrotic tissue lining the cavity itself, but nowhere in the lung tissue itself, and
3. No caseation or typical tuberculous formation in the lung.

CASE IN WHICH POST-MORTEM WAS MADE

No. 6. STAGE III.

1 AND 2

Name C.B.S. Age 34 Sex Male Date 1919
 Address 1000 1st St. City San Francisco State Cal.



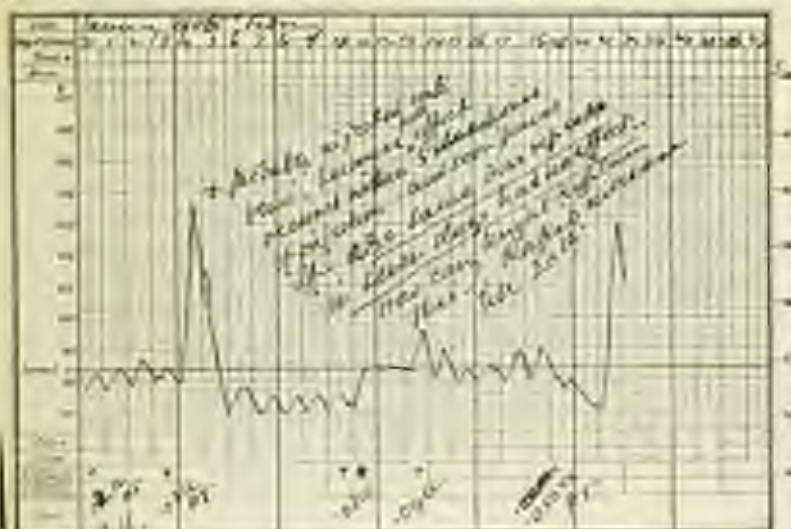
Name C.B.S. Age 34 Sex Male Date 1919
 Address 1000 1st St. City San Francisco State Cal.



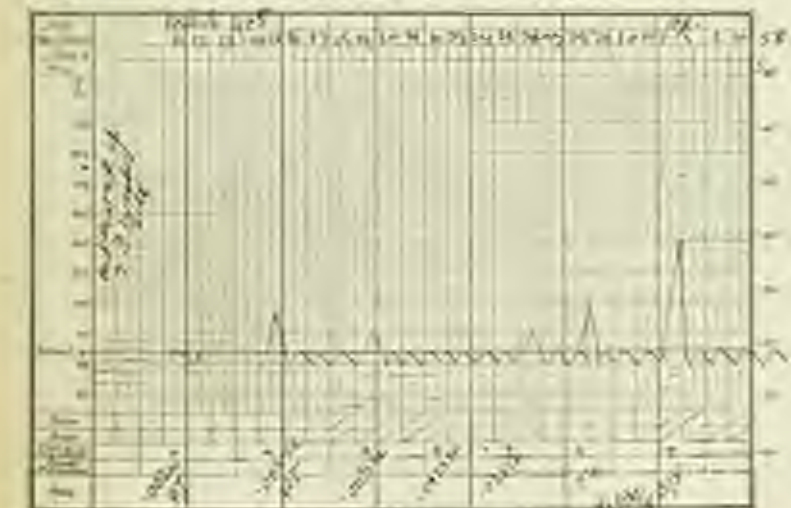
No. 6 STAGE III.

3 AND 4

Time _____ Date _____
 Station _____

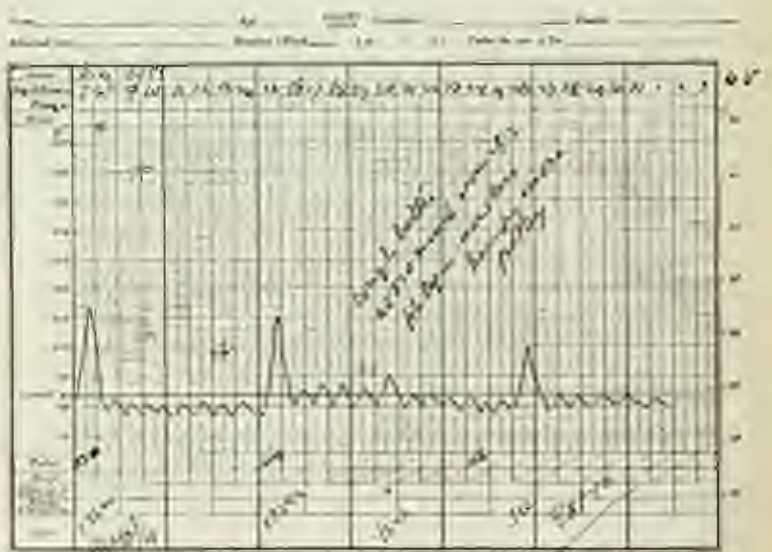
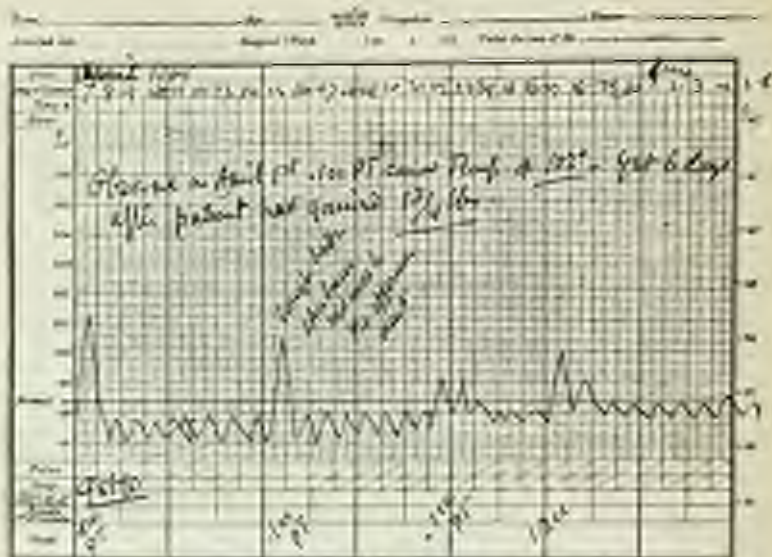


Time _____ Date _____
 Station _____



No. 6. STAGE III.

S. AND S.



No. 6 STAGE III.

7 AND 8

Time _____ Date _____
 Name _____

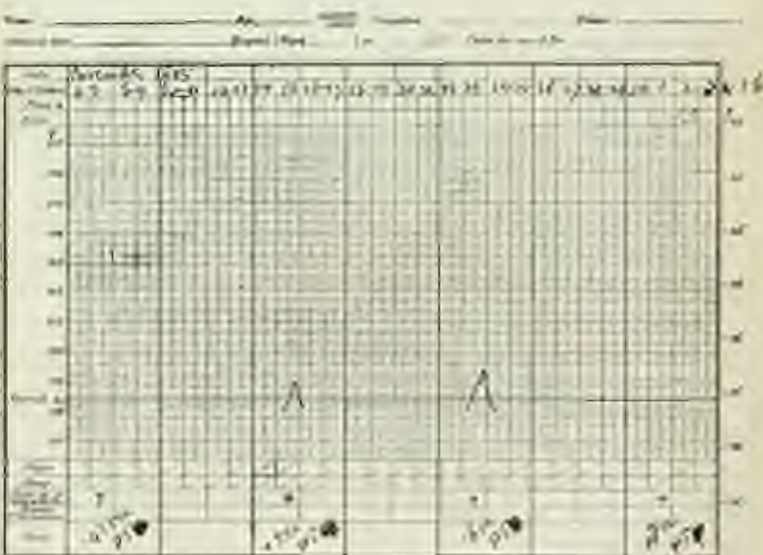


Time _____ Date _____
 Name _____



No. 6 STAGE III.

Q AND 80



No. 6. STAGE III.

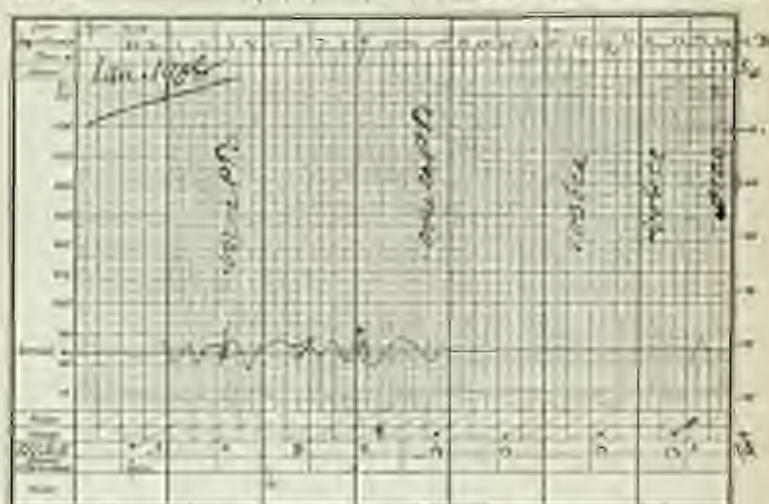
13 AND 14



NO. 5. STAGE III.

87 AND 88

Page No. 1007 Page No. 1008
 Date 10/10/1906
 Project (No.) 1007



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 Date 10/10/1906
 Project (No.) 1007



No. 6 STAGE III

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185 | 186 | 187 | 188 | 189 | 190 | 191 | 192 | 193 | 194 | 195 | 196 | 197 | 198 | 199 | 200 | 201 | 202 | 203 | 204 | 205 | 206 | 207 | 208 | 209 | 210 | 211 | 212 | 213 | 214 | 215 | 216 | 217 | 218 | 219 | 220 | 221 | 222 | 223 | 224 | 225 | 226 | 227 | 228 | 229 | 230 | 231 | 232 | 233 | 234 | 235 | 236 | 237 | 238 | 239 | 240 | 241 | 242 | 243 | 244 | 245 | 246 | 247 | 248 | 249 | 250 | 251 | 252 | 253 | 254 | 255 | 256 | 257 | 258 | 259 | 260 | 261 | 262 | 263 | 264 | 265 | 266 | 267 | 268 | 269 | 270 | 271 | 272 | 273 | 274 | 275 | 276 | 277 | 278 | 279 | 280 | 281 | 282 | 283 | 284 | 285 | 286 | 287 | 288 | 289 | 290 | 291 | 292 | 293 | 294 | 295 | 296 | 297 | 298 | 299 | 300 | 301 | 302 | 303 | 304 | 305 | 306 | 307 | 308 | 309 | 310 | 311 | 312 | 313 | 314 | 315 | 316 | 317 | 318 | 319 | 320 | 321 | 322 | 323 | 324 | 325 | 326 | 327 | 328 | 329 | 330 | 331 | 332 | 333 | 334 | 335 | 336 | 337 | 338 | 339 | 340 | 341 | 342 | 343 | 344 | 345 | 346 | 347 | 348 | 349 | 350 | 351 | 352 | 353 | 354 | 355 | 356 | 357 | 358 | 359 | 360 | 361 | 362 | 363 | 364 | 365 | 366 | 367 | 368 | 369 | 370 | 371 | 372 | 373 | 374 | 375 | 376 | 377 | 378 | 379 | 380 | 381 | 382 | 383 | 384 | 385 | 386 | 387 | 388 | 389 | 390 | 391 | 392 | 393 | 394 | 395 | 396 | 397 | 398 | 399 | 400 | 401 | 402 | 403 | 404 | 405 | 406 | 407 | 408 | 409 | 410 | 411 | 412 | 413 | 414 | 415 | 416 | 417 | 418 | 419 | 420 | 421 | 422 | 423 | 424 | 425 | 426 | 427 | 428 | 429 | 430 | 431 | 432 | 433 | 434 | 435 | 436 | 437 | 438 | 439 | 440 | 441 | 442 | 443 | 444 | 445 | 446 | 447 | 448 | 449 | 450 | 451 | 452 | 453 | 454 | 455 | 456 | 457 | 458 | 459 | 460 | 461 | 462 | 463 | 464 | 465 | 466 | 467 | 468 | 469 | 470 | 471 | 472 | 473 | 474 | 475 | 476 | 477 | 478 | 479 | 480 | 481 | 482 | 483 | 484 | 485 | 486 | 487 | 488 | 489 | 490 | 491 | 492 | 493 | 494 | 495 | 496 | 497 | 498 | 499 | 500 | 501 | 502 | 503 | 504 | 505 | 506 | 507 | 508 | 509 | 510 | 511 | 512 | 513 | 514 | 515 | 516 | 517 | 518 | 519 | 520 | 521 | 522 | 523 | 524 | 525 | 526 | 527 | 528 | 529 | 530 | 531 | 532 | 533 | 534 | 535 | 536 | 537 | 538 | 539 | 540 | 541 | 542 | 543 | 544 | 545 | 546 | 547 | 548 | 549 | 550 | 551 | 552 | 553 | 554 | 555 | 556 | 557 | 558 | 559 | 560 | 561 | 562 | 563 | 564 | 565 | 566 | 567 | 568 | 569 | 570 | 571 | 572 | 573 | 574 | 575 | 576 | 577 | 578 | 579 | 580 | 581 | 582 | 583 | 584 | 585 | 586 | 587 | 588 | 589 | 590 | 591 | 592 | 593 | 594 | 595 | 596 | 597 | 598 | 599 | 600 | 601 | 602 | 603 | 604 | 605 | 606 | 607 | 608 | 609 | 610 | 611 | 612 | 613 | 614 | 615 | 616 | 617 | 618 | 619 | 620 | 621 | 622 | 623 | 624 | 625 | 626 | 627 | 628 | 629 | 630 | 631 | 632 | 633 | 634 | 635 | 636 | 637 | 638 | 639 | 640 | 641 | 642 | 643 | 644 | 645 | 646 | 647 | 648 | 649 | 650 | 651 | 652 | 653 | 654 | 655 | 656 | 657 | 658 | 659 | 660 | 661 | 662 | 663 | 664 | 665 | 666 | 667 | 668 | 669 | 670 | 671 | 672 | 673 | 674 | 675 | 676 | 677 | 678 | 679 | 680 | 681 | 682 | 683 | 684 | 685 | 686 | 687 | 688 | 689 | 690 | 691 | 692 | 693 | 694 | 695 | 696 | 697 | 698 | 699 | 700 | 701 | 702 | 703 | 704 | 705 | 706 | 707 | 708 | 709 | 710 | 711 | 712 | 713 | 714 | 715 | 716 | 717 | 718 | 719 | 720 | 721 | 722 | 723 | 724 | 725 | 726 | 727 | 728 | 729 | 730 | 731 | 732 | 733 | 734 | 735 | 736 | 737 | 738 | 739 | 740 | 741 | 742 | 743 | 744 | 745 | 746 | 747 | 748 | 749 | 750 | 751 | 752 | 753 | 754 | 755 | 756 | 757 | 758 | 759 | 760 | 761 | 762 | 763 | 764 | 765 | 766 | 767 | 768 | 769 | 770 | 771 | 772 | 773 | 774 | 775 | 776 | 777 | 778 | 779 | 780 | 781 | 782 | 783 | 784 | 785 | 786 | 787 | 788 | 789 | 790 | 791 | 792 | 793 | 794 | 795 | 796 | 797 | 798 | 799 | 800 | 801 | 802 | 803 | 804 | 805 | 806 | 807 | 808 | 809 | 810 | 811 | 812 | 813 | 814 | 815 | 816 | 817 | 818 | 819 | 820 | 821 | 822 | 823 | 824 | 825 | 826 | 827 | 828 | 829 | 830 | 831 | 832 | 833 | 834 | 835 | 836 | 837 | 838 | 839 | 840 | 841 | 842 | 843 | 844 | 845 | 846 | 847 | 848 | 849 | 850 | 851 | 852 | 853 | 854 | 855 | 856 | 857 | 858 | 859 | 860 | 861 | 862 | 863 | 864 | 865 | 866 | 867 | 868 | 869 | 870 | 871 | 872 | 873 | 874 | 875 | 876 | 877 | 878 | 879 | 880 | 881 | 882 | 883 | 884 | 885 | 886 | 887 | 888 | 889 | 890 | 891 | 892 | 893 | 894 | 895 | 896 | 897 | 898 | 899 | 900 | 901 | 902 | 903 | 904 | 905 | 906 | 907 | 908 | 909 | 910 | 911 | 912 | 913 | 914 | 915 | 916 | 917 | 918 | 919 | 920 | 921 | 922 | 923 | 924 | 925 | 926 | 927 | 928 | 929 | 930 | 931 | 932 | 933 | 934 | 935 | 936 | 937 | 938 | 939 | 940 | 941 | 942 | 943 | 944 | 945 | 946 | 947 | 948 | 949 | 950 | 951 | 952 | 953 | 954 | 955 | 956 | 957 | 958 | 959 | 960 | 961 | 962 | 963 | 964 | 965 | 966 | 967 | 968 | 969 | 970 | 971 | 972 | 973 | 974 | 975 | 976 | 977 | 978 | 979 | 980 | 981 | 982 | 983 | 984 | 985 | 986 | 987 | 988 | 989 | 990 | 991 | 992 | 993 | 994 | 995 | 996 | 997 | 998 | 999 | 1000 | 1001 | 1002 | 1003 | 1004 | 1005 | 1006 | 1007 | 1008 | 1009 | 1010 | 1011 | 1012 | 1013 | 1014 | 1015 | 1016 | 1017 | 1018 | 1019 | 1020 | 1021 | 1022 | 1023 | 1024 | 1025 | 1026 | 1027 | 1028 | 1029 | 1030 | 1031 | 1032 | 1033 | 1034 | 1035 | 1036 | 1037 | 1038 | 1039 | 1040 | 1041 | 1042 | 1043 | 1044 | 1045 | 1046 | 1047 | 1048 | 1049 | 1050 | 1051 | 1052 | 1053 | 1054 | 1055 | 1056 | 1057 | 1058 | 1059 | 1060 | 1061 | 1062 | 1063 | 1064 | 1065 | 1066 | 1067 | 1068 | 1069 | 1070 | 1071 | 1072 | 1073 | 1074 | 1075 | 1076 | 1077 | 1078 | 1079 | 1080 | 1081 | 1082 | 1083 | 1084 | 1085 | 1086 | 1087 | 1088 | 1089 | 1090 | 1091 | 1092 | 1093 | 1094 | 1095 | 1096 | 1097 | 1098 | 1099 | 1100 | 1101 | 1102 | 1103 | 1104 | 1105 | 1106 | 1107 | 1108 | 1109 | 1110 | 1111 | 1112 | 1113 | 1114 | 1115 | 1116 | 1117 | 1118 | 1119 | 1120 | 1121 | 1122 | 1123 | 1124 | 1125 | 1126 | 1127 | 1128 | 1129 | 1130 | 1131 | 1132 | 1133 | 1134 | 1135 | 1136 | 1137 | 1138 | 1139 | 1140 | 1141 | 1142 | 1143 | 1144 | 1145 | 1146 | 1147 | 1148 | 1149 | 1150 | 1151 | 1152 | 1153 | 1154 | 1155 | 1156 | 1157 | 1158 | 1159 | 1160 | 1161 | 1162 | 1163 | 1164 | 1165 | 1166 | 1167 | 1168 | 1169 | 1170 | 1171 | 1172 | 1173 | 1174 | 1175 | 1176 | 1177 | 1178 | 1179 | 1180 | 1181 | 1182 | 1183 | 1184 | 1185 | 1186 | 1187 | 1188 | 1189 | 1190 | 1191 | 1192 | 1193 | 1194 | 1195 | 1196 | 1197 | 1198 | 1199 | 1200 | 1201 | 1202 | 1203 | 1204 | 1205 | 1206 | 1207 | 1208 | 1209 | 1210 | 1211 | 1212 | 1213 | 1214 | 1215 | 1216 | 1217 | 1218 | 1219 | 1220 | 1221 | 1222 | 1223 | 1224 | 1225 | 1226 | 1227 | 1228 | 1229 | 1230 | 1231 | 1232 | 1233 | 1234 | 1235 | 1236 | 1237 | 1238 | 1239 | 1240 | 1241 | 1242 | 1243 | 1244 | 1245 | 1246 | 1247 | 1248 | 1249 | 1250 | 1251 | 1252 | 1253 | 1254 | 1255 | 1256 | 1257 | 1258 | 1259 | 1260 | 1261 | 1262 | 1263 | 1264 | 1265 | 1266 | 1267 | 1268 | 1269 | 1270 | 1271 | 1272 | 1273 | 1274 | 1275 | 1276 | 1277 | 1278 | 1279 | 1280 | 1281 | 1282 | 1283 | 1284 | 1285 | 1286 | 1287 | 1288 | 1289 | 1290 | 1291 | 1292 | 1293 | 1294 | 1295 | 1296 | 1297 | 1298 | 1299 | 1300 | 1301 | 1302 | 1303 | 1304 | 1305 | 1306 | 1307 | 1308 | 1309 | 1310 | 1311 | 1312 | 1313 | 1314 | 1315 | 1316 | 1317 | 1318 | 1319 | 1320 | 1321 | 1322 | 1323 | 1324 | 1325 | 1326 | 1327 | 1328 | 1329 | 1330 | 1331 | 1332 | 1333 | 1334 | 1335 | 1336 | 1337 | 1338 | 1339 | 1340 | 1341 | 1342 | 1343 | 1344 | 1345 | 1346 | 1347 | 1348 | 1349 | 1350 | 1351 | 1352 | 1353 | 1354 | 1355 | 1356 | 1357 | 1358 | 1359 | 1360 | 1361 | 1362 | 1363 | 1364 | 1365 | 1366 | 1367 | 1368 | 1369 | 1370 | 1371 | 1372 | 1373 | 1374 | 1375 | 1376 | 1377 | 1378 | 1379 | 1380 | 1381 | 1382 | 1383 | 1384 | 1385 | 1386 | 1387 | 1388 | 1389 | 1390 | 1391 | 1392 | 1393 | 1394 | 1395 | 1396 | 1397 | 1398 | 1399 | 1400 | 1401 | 1402 | 1403 | 1404 | 1405 | 1406 | 1407 | 1408 | 1409 | 1410 | 1411 | 1412 | 1413 | 1414 | 1415 | 1416 | 1417 | 1418 | 1419 | 1420 | 1421 | 1422 | 1423 | 1424 | 1425 | 1426 | 1427 | 1428 | 1429 | 1430 | 1431 | 1432 | 1433 | 1434 | 1435 | 1436 | 1437 | 1438 | 1439 | 1440 | 1441 | 1442 | 1443 | 1444 | 1445 | 1446 | 1447 | 1448 | 1449 | 1450 | 1451 | 1452 | 1453 | 1454 | 1455 | 1456 | 1457 | 1458 | 1459 | 1460 | 1461 | 1462 | 1463 | 1464 | 1465 | 1466 | 1467 | 1468 | 1469 | 1470 | 1471 | 1472 | 1473 | 1474 | 1475 | 1476 | 1477 | 1478 | 1479 | 1480 | |
|---|---|---|---|---|---|---|---|---|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-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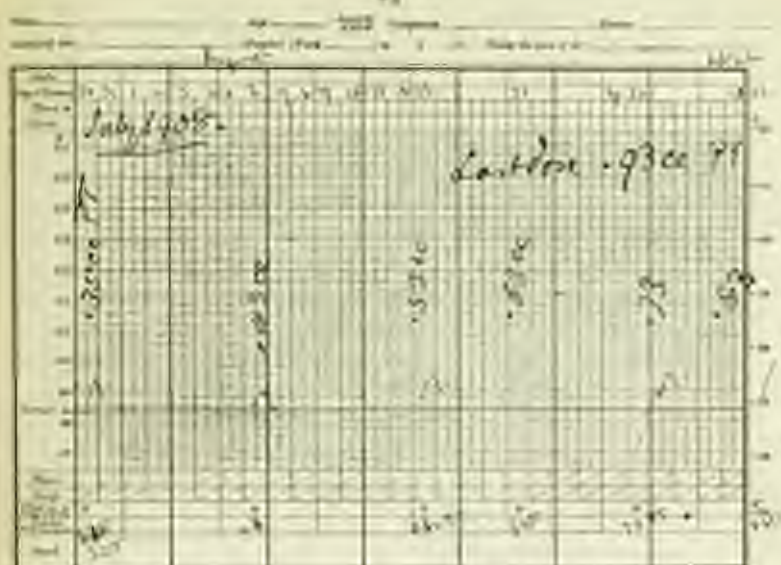
No. 6. STAGE III.

ET ASD 22



NO. 6. STAGE III.

#1



| 1 | 2 | 3 | 4 | 5 |
|----|----|----|----|---|
| 5% | 35 | 41 | 17 | 2 |

BLOOD EXAMINATION

| Case | Date | Time | General Observed Blood | Hb. (gms. per 100 c.c.) | Ht. (cm.) | Erythrocytes (per c.c.) | Variation in Leucocytes | | | | Latter's Count | | | | | Remarks |
|--------------------------------------------------------------|-----------|------------|--------------------------------------------|-------------------------|-----------|-------------------------|-------------------------|------|-----|-----|----------------|------|------|-----|-----|--------------------------------------------------------------------------------------------------------|
| | | | | | | | P | M | N | E | 1 | 2 | 3 | 4 | 5 | |
| G. H. S. (now with post mortem)
Case 6, Stage III | 14/xi/07 | 11 a.m. | Good colour and consistency | | | 3,450 | 78 | 19 | 2 | 1 | 24.5 | 39 | 31 | 6 | 0.5 | No tubercula since month. |
| | 11/x/08 | 12.30 p.m. | Very good col. and consistency | | | 3,800 | 69 | 27 | 4 | — | 31 | 37 | 23 | 8 | 1 | 0.13 cc. P.T.; about 1 month ago. |
| | 20/x/08 | 5.30 p.m. | Thick, dark, does readily | 3,120,000 | 100 | 12,500 | 74 | 33 | 1 | — | 15 | 39 | 38 | 16 | — | No tubercula for 3 or 4 months; wks. of feet; slight cyanosis. |
| Q... ..
Case 40, Stage II
in printed letters
above. | 4/v/06 | 12 m. | | | | 8,700 | 62 | 33 | 3 | — | 36 | 42 | 27 | 3 | — | Tubercula 3 days ago. |
| | 5/v/06 | 4 p.m. | | | | 6,900 | 75 | 21 | 1 | — | 34 | 45 | 26 | 3 | — | Tubercula 20 hours earlier; blood examined during reaction, T. 10° |
| | 25/v/06 | 11.30 a.m. | | | 80 | 8,400 | 65 | 32 | 1 | 2 | 31 | 39 | 34 | 0.5 | 0.6 | Last tubercula on 17th; reaction to 10° 4. |
| | 26 | 11.30 a.m. | | | 85-90 | 8,700 | 19 | 35 | 2 | 1 | 29 | 41 | 32.5 | 1 | 0.5 | Tubercula previous evening. |
| | 1/v/06 | 11.45 a.m. | | | | 8,600 | 78 | 17 | 3 | 1 | 27 | 44 | 32 | 5 | — | Last tubercula 30th. |
| | 4/v/06 | 12.30 p.m. | | | | 10,400 | 68 | 25.5 | 3 | 0.5 | 33 | 45 | 31 | 5 | 1 | Last tubercula 2nd. |
| | 7 | 11.50 | | | | 7,800 | 75 | 23 | 2 | 1 | 30 | 47 | 19 | 4 | — | Last tubercula 2nd, 5 days earlier. |
| | 9 | 10.45 | | | | 9,900 | 50 | 18 | 2 | — | 35 | 44 | 19 | 3 | — | 20 hours after tubercula. |
| | 13 | 11.30 | | | | 9,400 | 66 | 23 | 2.5 | 0.5 | 23 | 42 | 28 | 0.5 | 0.5 | Last tubercula 8th, 5 days earlier. |
| | 23/vii/ | 11.30 | | | | 7,800 | 68 | 33 | 3.5 | 0.5 | 32 | 45 | 30 | 3 | — | Last tubercula 16th; slight nasal catarrh; no fever. |
| | 22/xi/ | 11 a.m. | | | | 7,800 | 56 | 40.5 | 3 | 0.5 | 6.5 | 35.5 | 39.5 | 18 | 3.5 | Last tubercula (P.T. l.c.c.) on 22/x/06; patient feeling very well; gained 1 lb. weight during course. |
| | 11/xi/06 | | Good colour and consistency, also readily. | | | 8,800 | 47.5 | 44 | 4 | 0.5 | 13 | 24 | 37 | 12 | 3 | No tubercula since 22/x/06. |
| | 21/x/07 | 12 m. | | | | 5,900 | 82 | 37 | 1 | — | 7 | 39 | 43 | 18 | 2 | Tested early in March; reacted (92.5) to 0.03 cc. old T. |
| | | | | | | | | | | | | | | | | 27/xi to 14/iv/07; streng. mixed info. (T. 100°) |
| | 18/iv/07 | 1.25 p.m. | Before luncheon. A little thin and pale | | | 9,600 | 78 | 17 | 3 | — | 11 | 42 | 38 | 9 | — | Treated with T.R. 21/iv/07 to 28/vi/07. (2 v.c.) |
| | 23/vii/07 | 12.15 | Good colour and consistency | 5,480,000 | 50/100 | 8,200 | 82 | 28 | 3 | 1 | 12 | 40 | 35 | 10 | 2 | Gained 11 lbs. weight. |
| | 5/x/08 | 8.30 | Good colour and consistency | | 100 | 5,400 | 53 | 42 | 3 | 1 | 10 | 35 | 39 | 13 | 3 | Gained 16 lbs. more (88 to 104) |
| | 25/x/08 | 12 m. | Good colour and consistency | 3,500,000 | 95 | 2,000 | 46 | 50.5 | 3 | 0.5 | 8 | 33 | 41 | 15 | 2 | (Was tested 6/x/08, 0.03 cc. T. without reaction.) |

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BLOOD EXAMINATION—Continued

| Patient | Date | Time | General Condition & Notes | Erythrocytes
(per c.c.) | Hb. Gm. | Leucocytes
(per c.c.) | Differential of Leucocytes | | | | Sedimentation Rate | | | | | Remarks |
|------------------------------------------|----------|-----------|----------------------------------------------|----------------------------|---------|--------------------------|----------------------------|------|-----|-----|--------------------|------|------|------|-----|---------------------------------------------------------------------------------------------------------------------------------------------------|
| | | | | | | | T.B.C. | L. | S. | M. | 1 | 2 | 3 | 4 | 5 | |
| Mrs. G.
(pt. 28)
Case 72, Stage I | 11/11/36 | | Thin, pale, does very freely | 3,350,000 | 40 | 15,400 | 54 | 12 | 2 | — | 12 | 30 | 31 | 18 | 1 | Continued 7½ days earlier; T. 99°; no pelvic infection. |
| | 3/10/36 | 11.45 | | | 80 | 8,000 | 73 | 20 | — | — | 3 | 28 | 44 | 21 | 4 | Slight polydipsia; no pyrexia. |
| | 18/10/36 | 9.30 | Thin, pale, does freely, readily coagulates. | 4,580,000 | 40 | 4,100 | 54 | 24 | 2 | 2 | 45.5 | 30.5 | 27 | 8 | 3 | Slight weight; no tuberculin test; a few hours later was rejected with worn old T. reacted to 1000, and on treatment with T.R. began on 22/10/36. |
| | 18/10/36 | | | | | | | | | | | | | | | |
| | 18/12/36 | | Thin, pale, slight pyrexia. | 3,560,000 | 30 | 4,700 | 54.5 | 40 | 1 | 1.2 | 19.5 | 30.5 | 34 | 16.5 | 1.5 | Last tuberculin 3 days ago, reacted to 1000 P.P.T.B. 18 mm. Rejected vaccine; greatly improved in health but no gain of weight. |
| | 20/11/37 | 11 a.m. | (absent) | | | | | | | | | | | | | |
| | 16/11/37 | 4 p.m. | 24 hrs after T.R. 100 cc. | | | 7,500 | 72.5 | 27 | — | 0.5 | 8 | 20 | 45 | 15 | 3 | Shocked infant for 3 months. |
| Mrs. G.
(pt. 29)
Case 82, Stage I | 21/10/37 | 11.30 | Thin. | 4,280,000 | 30 | 8,300 | 42 | 22.5 | 3 | 0.5 | 7 | 35 | 45 | 30 | 2 | Feels much better still. |
| | 24/11/37 | 11.30 | ? Thin, good colour. | 4,190,000 | 30 | 4,300 | 58 | 34.5 | 3 | 0.5 | 7 | 35 | 43 | 18 | 1 | Very well, gained 20 lbs. weight in 1½ years. |
| | 2/12/37 | 8.15 p.m. | Good colour and consistency | 4,000,000 | 30 | 7,350 | 47.5 | 30 | 0.5 | — | 24 | 44 | 38 | 2 | — | |
| | 22/10/37 | 10 a.m. | Good colour and consistency | 4,800,000 | 30 | 4,000 | 57 | 38 | 2 | 1 | 15 | 37 | 44 | 4 | — | Last tuberculin 22 hours earlier; no reaction. |
| | 7/11/38 | | | | | | 57 | 37 | 2 | — | 15 | 36 | 44 | 4 | 1 | Some months after last tuberculin; very well. |
| | 21/12/38 | 11 a.m. | Good colour and consistency | 4,140,000 | 40 | 16,000 | 73 | 35 | 2 | 1 | 45 | 46 | 8 | — | — | 0 3rd stage; positive; streptococcal mixed infection at lung; no tuberculin. |
| | 22/1/39 | 11 a.m. | | | | 10,000 | 55 | 33 | 2 | — | 42 | 45 | 18 | 1 | — | 26 hours after maternal report. |
| Mrs. G.
(pt. 30)
Case 30, Stage II | 9/12/36 | 8.30 | | 4,300,000 | 40 | 12,000 | 70 | 22 | 0.5 | 0.5 | 25.5 | 47 | 30 | 2 | 0.5 | ♀ 12. dying; streptococcal; no tuberculin. |
| | 2/12/36 | 11.30 | Good colour and consistency | 4,120,000 | 40 | 10,400 | 40 | 12 | 1 | — | 51 | 40.5 | 8 | 0.5 | — | ♀ 12. S. dying; no tuberculin. |
| | 9/1/37 | 10.30 | | 4,044,000 | 30 | 8,600 | 73 | 28 | 1 | — | 51 | 45 | 12.5 | 0.5 | — | ♀ 12. S. dying; had a little tuberculin. |
| | 1/2/37 | 11.30 | | 4,400,000 | 34 | 10,800 | 32 | 8 | — | — | 32 | 41 | 19 | 4 | 1 | ♀ 12. dying; had a little tuberculin. |
| | 20/11/37 | 11 a.m. | Thick, very good colour | 4,000,000 | 30 | 16,500 | 72 | 37.5 | 0.5 | — | 45 | 38 | 18 | 2 | 1 | ♀ 12. dying. |
| | 5/12/37 | 9.30 a.m. | Thin and pale. | 4,800,000 | 35 | 10,400 | 47 | 30.5 | — | 0.5 | 44 | 41 | 33 | — | — | ♀ 12.; last injection 10 days earlier; T. to 100°. |
| | 25/12/37 | | | 4,500,000 | 40 | 14,200 | 41 | 18 | 1 | — | 37 | 41 | 28 | 8 | 1 | ♀ strept. infection of lung and elsewhere. |
| Mrs. W.
Case 33, Stage II-D | 10/11/36 | 11.30 | | 3,900,000 | 30 | 14,400 | 48 | 31 | 0.5 | 0.5 | 42 | 43 | 14 | 0.5 | 0.5 | |
| | 28/11/37 | 3 p.m. | | | 30 | 12,400 | 70 | 31 | — | — | 35 | 42 | 35 | 3 | — | After TOA (1000) P.T.O. (1000) and 100 cc. T.R. very sensitive—gained 21 lbs. |
| Mr. C.
Case 30, Stage II | 14/11/37 | | Good colour and consistency | | | | 41 | 34 | 2 | 1 | 11 | 34 | 41 | 11 | 1 | Opsonic index 0.52. 100 cc. P.T. Year ago. |
| | 7/10/38 | 10 a.m. | | | 30 | | 36 | 31 | 2 | — | 24 | 41 | 28 | 4 | 1 | 2 to 3 weeks after last dose of P.T. given. |
| D. K. (pt. 13) | 18/10/38 | 10 a.m. | | 3,100,000 | 30 | 7,200 | 70 | 24 | 2 | — | 47 | 40.5 | 12 | 0.5 | — | Little girl treated by F.G.G. |
| | 2/11/38 | 10.30 | | | 30 | 14,300 | 78.5 | 30 | 1.5 | — | 35 | 41 | 18 | 3 | — | 17 days after last injection of tuberculin. |
| | 8/11/38 | 10.15 | | 3,810,000 | 30 | 8,000 | | | | | 36 | 40 | 27 | 4 | — | |
| | 28/11/37 | 11 a.m. | | 3,200,000 | 30 | 18,000 | 41 | 14 | 2 | — | 23 | 41 | 25 | 4 | — | 14 days after 1000 cc. T.R. reaction, which produced no reaction. Patient continued to improve until last blood at 41 and of 2800. |

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PART VI

THE TECHNIQUE OF TUBERCULIN TREATMENT

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page 245*

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PART VI

TECHNIQUE

THERE are many forms of tuberculin. While there is a consensus of opinion in favour of Old Tuberculin as the best agent in diagnosis, there is not any agreement as to either the best form of tuberculin to be used as a remedy for the disease, or the method of its administration. For practical purposes we may distinguish between the forms derived from tubercle bacilli of the human type and those derived from bacilli of the bovine type. Again, apart from this distinction, we may broadly classify tuberculins in two groups: those (exotoxins) which are mere filtered extracts of tubercle bacilli and those (endotoxins) which contain the less soluble substance of the micro-organisms. The former (extracts) also differ according to the process of extraction. Thus, Old Tuberculin is made from the same material as T.O.A., but under the influence of heat in the process of concentration certain substances enter the extract, and subsequently the filtrate, which do not pass into the simple extract of T.O.A. Accordingly, Old Tuberculin is not merely a concentrated T.O.A., but thus reduced by heat to one-tenth of the bulk of T.O.A. is *about fifty times more powerful* in its effects. The same applies to P.E. and P.T.O. These are matters of the greatest importance if the methods which I have adopted are in the main followed. No doubt the effects of the extracts which consist essentially of exotoxins are different from those of the emulsions which contain not only some exotoxins, but mainly the endotoxins which are intimately united to the solid cell substance. Experience has to show which of these various preparations are the better agents in stimulating living cell elements to produce

the immunising substances to which we must look for the beneficial results of specific treatment. Even did we know which were the more powerful immunising agents, we have still to learn how to reach the highest degree of active immunisation. At present there are the two extreme schools: the one claiming the best results by extremely small doses, the other claiming brilliant results by extremely large doses.

It may be well here to point out the unwisdom of describing the amount of a dose by an arbitrary and erroneous system. It is infinitely better to retain the simple uniform system of measurement which can be applied to all forms of tuberculin, no matter what the method of preparation may have been. Certainly in the two preparations (T.E. and P.T.E.) it is possible to estimate the dose according to the weight of dead tubercle bacilli used in making the emulsion. Thus 1 cc. of T.E. does contain 5 mgs. of pulverised tubercle bacilli, and therefore it is possible to calculate the dose in relation to the weight of dead tubercle bacilli; but in such a case one should speak of the dose as a certain quantity of the dead bacilli. Thus:—

1 cc. of T.E. = 5 mg. dead tubercle bacilli;

∴ 0.01 cc. of T.E. = 0.05 mg. dead tubercle bacilli;

or $\frac{1}{100}$ cc. of T.E. = $\frac{1}{20}$ mg. of dead tubercle bacilli;

but throughout medical literature one finds this latter dose described as $\frac{1}{10}$ mg. of T.E. It is nothing of the kind. With regard to the other preparations of tuberculin, such as T.R., T.O.A., Old Tuberculin, P.T.O., P.T., etc., this system of describing the amount of the dose is erroneous and should be absolutely abandoned. There is no difficulty in measuring the doses in fractions of a cubic centimetre, and in a system in which progressive dosage is practised (by which I mean a series of doses in which each subsequent dose has a definite relation to the previous dose), it is extremely important to use a method of measurement which can be easily understood and requires no correction of errors.

One can only measure the preparations T.O.A., T.A. (Old Tuberculin), P.T.O., and P.T. according to their bulk, and the profession must sooner or later discard the erroneous

system of measuring doses and describing them which we owe to an inaccurate knowledge of the composition of various preparations of tuberculin which are now being so freely used in every country. Everyone knows what a cc., $\frac{1}{2}$ cc., or $\frac{1}{10}$ cc., or $\frac{1}{100}$ cc., etc., means, and in preparing these doses we shall have to contend with quite enough errors of our own which are difficult to avoid, without strewn our path with gratuitous errors that can and should be eliminated at the outset. In fact, the system of dosage in vogue at the present time should be now and for ever abandoned.

Further, in the administration of tuberculin the medical profession may with impunity cast off the unnecessary burden of determining the opsonic index. The graduation of doses in accordance with Wright's interpretation of the opsonic index is not only fantastic and worthless, but may be a serious hindrance to the better use of the remedy. Perhaps the opsonic index will throw some light upon the curious phenomenon of over-sensitiveness (anaphylaxis), but as a practical guide in the choice of doses so strongly recommended by Wright it is faulty, flimsy, and futile. The clinical experience of a well-trained physician is a far better guide than these methods of the laboratory with a semblance of truth in them. Therefore, a further prelude to the proper use of tuberculin is that the opsonic index as a guide in the choice of doses should be ignored, if not abandoned. I am able to show also by scores of records that the "negative phase" of Wright is but another bogey in the way of success. Instead of following Wright's system of spacing out doses for seven, ten, fourteen days, it is best to give doses every three, four, five days, unless there are obvious clinical contraindications.

The site of the injection is not important, provided one avoids the site of large blood-vessels, especially the large superficial veins. Injected into a vein the effect is far more rapid and more severe, and the local swelling and infiltration fall altogether. A rigor generally ushers in the severe reaction, and temperature may reach 104° or 105° . I generally inject into the upper arm on the outer side—though

one may inject into the lower arm. Patients can inject into the outer side of their thigh. For large doses the region between the shoulder-blades or under and to the outer side of the lower angle of this bone are convenient—especially when T.R. or T.E. is given.

The most important factors in determining the amount of increase, and the frequency, of the doses are disturbances of temperature, general disturbance, indicated by loss of appetite, malaise, sense of weakness, etc., and loss of weight. A slight rise of temperature, say to $99\frac{1}{2}^{\circ}$, suggests caution in the increase of the dose, and it is better perhaps to wait until the temperature has been normal for three days after this rise. If the temperature be 100° , one may give the same dose in about five days, or sometimes a slightly larger dose. In the first month of treatment the temperature is more easily disturbed than subsequently. Then it is wise not to increase the doses if the temperature be above 100° . If the temperature be raised to 101° the same dose as the previous one may be given, but only after an interval of six days. If the temperature be 102° , the previous dose should be repeated in seven days. If the temperature be above 102° , it may save time to diminish the dose and repeat it every three or four days until the temperature is not affected by it. Sometimes by repeating this smaller dose every second day for a fortnight the sensitiveness to even much larger doses is obviated¹. With these general indications one is able to increase doses until at intervals of three, four, or more days very large doses are well tolerated. The early doses, no matter how small, are likely to cause a rise of temperature.

In an ordinary adult I generally begin with $\frac{1}{10}$ to $\frac{1}{8}$ cc. of P.T.O., but one may begin with $\frac{1}{10}$ to $\frac{1}{20}$ cc. If no rise of temperature occurs the doses may be rapidly increased, certainly by $\frac{1}{2}$ or $\frac{1}{3}$ or even $\frac{1}{2}$ of the previous dose. Thus by slow degrees the dose is increased till 1 cc. of P.T.O. is given. The larger dose of P.T.O. sometimes causes a good deal of pain for two or three minutes after the injection, but this passes off, and otherwise has no objection.

¹ See record of temperature, p. 294 (Mr. Tiley's case).

Then one proceeds to the stronger preparation, P.T. Now P.T. is about fifty times as strong as P.T.O., so that after 1 cc. of P.T.O. has been given, the first dose of P.T. should be about 0.02 cc. or $\frac{1}{50}$ cc. These doses are again given at intervals of three, four, five days, provided there be no rise of temperature, until 1 cc. of P.T. is injected at a dose. P.T. causes very little trouble at the site of injection, but is more likely to cause a rise of temperature than the later doses of P.T.O. However, acting on the same general lines in increasing the dose and varying the interval of time according to the degree of fever during the reaction, there are no serious difficulties. Always, however, great care is necessary if a patient complains of lassitude, weakness, depression, especially *when there is also some loss of weight*. A loss of weight after a rise of temperature is sure to occur, but if subsequently the original weight is restored and a pound or two is added this is a favourable sign. In fact, gain in weight which is not due to over-feeding or a life of indolence or idleness is one of the best of signs. Mitulescu's work proves that under the influence of tuberculin treatment the process of catabolism is checked, so that there is less loss of phosphorus and nitrogen to the body. The gain in weight is only another view of the same phenomenon.

Measurement of Small Doses.

In this method of progression from weaker to stronger preparations, e.g. from P.T.O. to P.T. and thence to Old T. (T.A.), there is no necessity to make dilutions except for the first few doses. Thus in order to measure $\frac{1}{100}$ cc. of P.T.O. one has merely to obtain glass pipettes of a somewhat fine calibre. (For this purpose I find Mace and Haldane's glass droppers very convenient. They are very cheap and can be readily sterilised in a spirit-lamp.) I measure off $\frac{1}{10}$ cc. in these pipettes. This is readily done by means of Koch's syringes containing 1 cc., and a mark is made with ink along the pipette at a point which measures $\frac{1}{10}$ cc. Having then this pipette marked as $\frac{1}{10}$ cc. one may subdivide

it again into tenths, so that in this simple way one can measure $\frac{1}{10}$ cc. Now, if one has already made a dilution of the P.T.O. in the strength of 1 in 10, then an amount up to $\frac{1}{10}$ of the length of this column on the pipette is equivalent to $\frac{1}{1000}$ cc. I even go further, and mark the doses on the chart according to the length of the dose in this pipette of fine calibre, because, after all, the important thing is that the doses in sequence should have a definite and easily recognisable ratio to one another. In this way it appears to me there is less risk of giving wrong doses than if several dilutions are used. The eye controls everything and becomes wonderfully accurate with a little training, so that as soon as the dose is measured on the chart by this graphic method it is difficult to see how a wrong dose can be given. This avoidance of a wrong dose is all the more important in a method which aims at a high degree of immunisation by means of very large doses. When small fractions of a milligram are injected at a dose there are not likely to be any serious effects, if twenty times the dose is given.

It goes without saying that the strictest asepsis must be carried out. The syringe must be boiled, the pipette must be sterilised, and every care taken in making dilutions and handling the fluid, that the preparations are kept perfectly sterile. The skin where the injection is given may be simply rubbed well with spirit or spirit and ether. I usually place plaster just sterilised in the flame upon the prick-mark as a precaution, but I have to record one instance of remarkable idiosyncrasy. The plaster used (Mead's Plaster) in this woman excited the most intense purulent Dermatitis, strictly limited to the area covered by the plaster. The pus dried and formed a hard scab, beneath which the skin continued to discharge for some weeks, and finally completely healed.

Perhaps one should emphasise the obvious importance of using every possible care to avoid the risk of giving a dose of P.T. instead of a dose of P.T.O. or a dose of Old T. (T.A.) when one meant to give P.T. It is not entirely superfluous to mention such possible mistakes, *locus humanum est errare*, and if such a mistake were made, by which P.T. was given

when P.T.O. was intended, the patient would be receiving fifty times as great a dose as was really intended. In tuberculin treatment by means of large doses care must always be exercised in the selection of the agents themselves.

The Technique of Tuberculin Treatment.

All methods of treating tuberculosis require time. It is now recognised that sanatorium treatment should be continued at least six months in the majority of cases, and too often this is insufficient. In tuberculin treatment also, one cannot expect good results unless one proceeds to large doses, and it takes at least six months to work up to these large doses. As I have already stated, I begin with P.T.O., then proceed to P.T., and finally use Old Tuberculin. If one begins with 0.01 cc. or 0.001 cc. and proceeds to a final dose of 1 cc. to 2 cc. of Old Tuberculin, the final dose is literally a quarter of a million to two million times as potent as the first dose. To reach this large dose one proceeds by gradual increases, avoiding, as far as possible, reactions much above 100°. To measure the small doses one requires a 1 cc. syringe, a pipette of fine calibre on which one can measure off $\frac{1}{10}$ cc. and the ordinary diluting fluid (normal saline, containing $\frac{1}{2}$ per cent carbolic acid). After marking with ink the point of the pipette, which measures $\frac{1}{10}$ cc., one passes the ink-mark through the flame to fix it. By my plan it is usually only necessary to make one dilution, that of P.T.O. in the strength of 1 in 10. This dilution of 1 in 10 is made by putting one part of P.T.O. in a sterilised watch glass and adding to it nine times the quantity of the diluting fluid (normal saline, with $\frac{1}{2}$ per cent carbolic acid). Then, in order to measure $\frac{1}{100}$ cc. of P.T.O., one merely takes in the pipette a column of this diluted fluid equal to $\frac{1}{10}$ part of the column, which represents $\frac{1}{100}$ cc. If one wishes to measure $\frac{1}{1000}$ cc. (0.001 cc.), one must make a first dilution of 1 in 100 instead of 1 in 10.

Koch's 1 cc. syringes are graduated in one-tenths—so that it is easy to graduate the pipettes by these syringes. As pipettes I always use Mace and Haldane's droppers. An

amount of the dilution already made (1 in 10) equal to $\frac{1}{10}$ of the slender column of this graduated pipette is $\frac{1}{1000}$ cc., sometimes called a milligram. I have found this method very handy, because one is able at once to record on the temperature charts the length of this column in a graphic way instead of recording the actual dose. In the system of progressive doses it is more important to maintain the ratio of successive doses than to record the amounts. By this system the eye always keeps under its control this accurate ratio. If the previous dose has had no effect, as judged by temperature, weight, and the general state of patient, one may increase the dose by $\frac{1}{2}$ or $\frac{1}{4}$ cc., if the patient has hitherto manifested sensitiveness, by $\frac{1}{4}$ or $\frac{1}{2}$. Of course, if the previous dose has caused a reaction, even as slight as 99.5°, it is wise merely to repeat the same dose in four days. If the reaction amounts to 100°, one repeats the same dose in five days. If the temperature be 100.5°, one repeats in six days. If 101° or more, one repeats the same dose in a week.

As soon as the dose of the dilution (1 in 10) is $\frac{1}{10}$ cc., then one may proceed at once to measure P.T.O. from the original solution. This amount is also measured in these slender Mace and Haldane droppers, and one begins at first with rather more than one-tenth of the measured length of the column of fluid in the pipette. One increases the dose then at three, four, five, etc., days' interval according to the rise of temperature until the amount can be readily measured in the syringe itself, and each dose should be accurately recorded on the temperature chart each time *before the dose is given*. The dose of P.T.O. is further increased until 1 cc. is given. Then we change to P.T., which is about fifty times as strong as P.T.O. Accordingly, the first dose of P.T. is $\frac{1}{50}$ cc., which can also readily be measured in a similar slender pipette, or dropper. The amount of P.T. necessary for this dose is about $\frac{1}{2}$ of the slender column of the pipette, which measures $\frac{1}{10}$ cc. Always in giving these small quantities one adds a sufficiency of diluting fluid to the measured dose in the syringe itself. This dose of P.T. is graphically recorded on the temperature

chart, and the progressive increases to be subsequently given may be accurately guessed. The dose of P.T. is increased until 1 cc. is taken without any effect. After P.T. Old Tuberculin is given, which is about five times as strong as P.T., so that, after giving 1 cc. of P.T., one proceeds to $\frac{1}{5}$ cc. of Old Tuberculin. Then, if possible, Old Tuberculin is gradually increased until 1 cc. is given. It may be better to use T.A.F. instead of T.A., but I have not had time to prove this.

I find it simpler to record the smaller doses by this graphic method, because it dispenses with the necessity of making a series of dilutions. It seems easier for me to avoid the risks of giving wrong doses in this way than in any other, and more than this, this system saves an enormous amount of time. Accordingly, all my early doses are recorded by a stroke or line made with the pen, which represents the exact length in the pipette of the column of fluid which has been given. No calculation is necessary. One uses the fluids in amounts represented by, in the first instance, the length of the column in the slender pipettes, and later by the amounts measured in the syringe itself.

Further, I prefer Koch's syringe to all others—so easy to sterilise, so easy for the direct and immediate measurement of the doses, so handy for this particular method of administration. But there is no virtue in this system above any other system. The virtue lies in the dose alone, and provided the ratio existing between the successive doses is maintained each man is at perfect liberty to choose his own method. I find that most medical men who have visited my tuberculin dispensary and watched my methods find them simple and straightforward. Further, when one is using other preparations, such as T.R. and T.E., which may be given in two and three centimetre doses, one can refill Koch's syringe without withdrawing the needle, if this be thought advisable.

The Administration of Tuberculin by the Mouth.

Although the investigations of Professor Koch and others have shown that tuberculins administered by the mouth were either destroyed by the gastric juice or inert in their effects when they had passed through the stomach, there have always been isolated observers who casually imagined and asserted that this form of administration was useful and free from any of the risks associated with its administration by subcutaneous injection. One of these observers, Dr. Arthur Latham, has persistently maintained that this method of administration yielded very successful results in his hands. It is well, therefore, to give an outline of the work which has been done at Koch's Institute under the guidance of Professor Gaffky and Professor Jochmann by Stabsarzt Dr. B. Möllers and Dr. Heinemann.

They used in their investigations the various preparations that are on the market, some of them containing the extracts, such as Old T., and some tubercle bacilli, finely divided. These preparations were made up in capsules so as to protect the contents from the action of the gastric juice. Drs. Möllers and Heinemann proved that some of the preparations passed through the stomach without injury, while in other cases the capsules were dissolved both in the stomach and in the intestines. It is therefore possible to enclose the tuberculins in capsules in such a way that the effect of the action of the gastric juice is eliminated, but this result was not always obtained. They next showed that Old T. submitted to the action of gastric juice was distinctly injured, while hydrochloric acid of the strength found in the gastric juice had very little effect. These tests were made by means of von Pirquet's reaction, by the method in vogue for standardising tuberculin preparations in guinea-pigs and by the fixation of the complement, and the results were the same; namely, that the gastric juice had an injurious effect upon tuberculin preparations and destroyed or damaged their specific action. By the fixation of the complement also it was shown that trypsin and combined pepsin and trypsin caused a much greater injury to

the antigen. Further experiments were made with the two main forms of tuberculin, the simple extract and the finely ground tubercle bacilli. Doses varying from 101 mg. up to even 100 mg. were administered by the mouth to tuberculous patients, and Professor Koch himself personally watched the experiments. Old T. was given in doses from 2 up to 250 mgs. (25 cc.). These doses were given in capsules and as many as 4 capsules, equivalent to 1 cc. Old T., were thus administered. The doses in these capsules were far in excess of the doses which were given by Dr. Arthur Latham and those who believe in this method. Some of these investigations, too, were made upon patients who had already been treated with one or other form of tuberculin.

In these investigations three chief points were considered; firstly, whether administration by the mouth (stomachal administration) possessed any diagnostic value; secondly, whether by these means any demonstrable degree of immunity was reached; and thirdly, whether there was any advantage in treating patients in this way. It will be observed, that the largest dose used, 100 mgs. of dead bacilli, was twenty times the dose contained in 1 cc. of T.E., the so-called "bacillary emulsion."

1. "Seeing that these immense doses administered by the mouth and stomach and intestinal tract to tuberculous persons failed in the great majority of cases to produce either a rise of temperature or any other sign of a reaction, we have far better proof than any theoretical assumption can provide, that this method of using tuberculin is absolutely unsuitable for diagnostic purposes" (*Deutsche Medizinische Wochenschrift*, No. 40, 1911, p. 1826).

2. The second point whether thus administered tuberculin preparations can produce any demonstrable degree of immunity is answered in just the same way. It has been proved that after a dose of Old Tuberculin, equivalent to about 100-300 mgs., has been administered subcutaneously in the ordinary course of treatment, von Pirquet's reaction fails, although it has been well marked at the outset. On the other hand, after administration by the mouth of much larger doses of Old T. or bacillary substance, von Pirquet's

reaction is just as marked as ever. In one case, at the end of treatment, a negative von Pirquet became positive. Out of many cases a weakening of the von Pirquet reaction was observed only thrice, while in no instance did a positive von Pirquet reaction become negative. Thus the *von Pirquet phenomenon* showed that there was no noteworthy degree of immunity in persons treated by the alimentary tract.

These observations also confirm the original observations of Koch, that after administration by the mouth the subcutaneous injection of tuberculin is just as obvious as when no tuberculin has been given. In one instance a patient reacted with high fever to 4 consecutive doses, by subcutaneous injection of .02 mg. bacillary emulsion, while the same preparation given within a month and a quarter in 10 portions totalling 536 mgs. (4 times 100 mgs. at each dose) was tolerated without the slightest reaction; yet a subsequent subcutaneous injection of .02 mg.—only $\frac{1}{2680}$ part of the dose administered by the stomach—again caused a rise of temperature to 39.5° C. (103° F.). Thus the sensitiveness of the organism to tuberculin had not been affected in the very least by a dose administered by the stomach, which was many thousand times bigger than the dose necessary to cause the subcutaneous reaction. In another case 4580 mgs. of Old T. given by the mouth did not render the patient insensitive to 5 mgs. (.005 cc.) of Albemarle Free Tuberculin. Another patient who had tolerated without reaction first of all 682 mgs. of pulverised tubercle bacilli and then 5660 mgs. of Old T., both by the mouth, reacted to 5 mgs. (.005 cc.) of Old T. with a temperature of nearly 103° F. Thus there is not much room for the view that tuberculin given by the mouth can produce any palpable degree of immunity. Certainly the majority of these cases gained weight. Can we have a better proof that in the treatment of tuberculosis in hospitals and in sanatoria, the gain in weight is not due to any improvement in the tuberculous process, but is merely an incidental effect of ordinary hospital treatment? I repeat here what I have many times observed, that gain in weight is but a poor standard for testing the value of treatment; but this gain in weight and subjective improve-

ment incidentally shows also that tuberculin given by the mouth, even in large doses, does no harm.

The conclusions of this inquiry are that the active principle of tuberculin is seriously impaired by pepsin and trypsin; secondly, tuberculous subjects highly sensitive to tuberculin may tolerate doses of 1 cc. of Old T. and 100 mg. of bacillary substance without any fever or other reaction; thirdly, tuberculin immunity is not induced by tuberculin administered through the alimentary tract; fourthly, for diagnostic purposes this method of administering tuberculin is absolutely unsuitable. For therapeutic purposes administration by the mouth has little or no useful effect because the active principle is damaged, absorption is difficult and defective, and the dosage is at best variable and uncertain. It is therefore to be hoped that those who have any regard, not merely for the credit of science, but also for the good of their patients, will continue to use the method which is simple, trustworthy and effective, and discard a method which has been proved to be unsuitable, untrustworthy, ineffective and unscientific. We may learn also how easy it is for observers with limited vision and no regard for logic to arrive at utterly wrong conclusions when they merely record the haphazard results of personal experience which are beyond the control of logical and scientific methods. It is surely high time that we should give a decent and final burial to the view that tuberculin given by the mouth is a reasonable and useful procedure—the mourners should be wiser if sadder men.

Choice of Tuberculin for Diagnosis and Treatment.

(a) DIAGNOSIS

The choice is easy enough in diagnosis. One uses mainly Old Tuberculin, or T.A.F., and in ordinary cases I prefer the original method of Koch to any of the later methods (von Pirquet's Skin Reaction; Calmette's Ophthalmic Reaction; or Moro's Percutaneous Reaction). Koch's original method may be used in all cases of doubt at any age in suitable doses, except when there is fever. When there is fever von Pirquet's

method should be used. It is sometimes interesting to compare the effects produced by different forms of tuberculin, especially Old Tuberculin (T.A.), and Perlsacht Tuberculin (P.T.), but as yet these experiments do not help us in the selection of the form of tuberculin for individual cases. I do not approve of Calmette's method, which has no advantage over Koch's method in fever-free cases and no advantage over von Pirquet's method in febrile cases. It has these distinct disadvantages: firstly, that it may excite severe inflammation of the eye, lasting days and even weeks, sometimes invading the cornea. And secondly, subsequent therapeutic doses injected under the skin are often followed by a fresh attack of conjunctivitis. This conjunctivitis may be started not only months but a year after the ophthalmic reaction has been exploited.³

(b) TREATMENT

My own experience has led me to adopt in a routine fashion the tuberculin of bovine strains in the preliminary stages of treatment. I generally begin with P.T.O., but I never end with it. P.T.O. is merely a means for preparing the way for P.T. With P.T. administered in large doses of 0.7 cc.—1 cc., one may obtain surprisingly brilliant results. In well-to-do persons whose surroundings are healthy and the food supply adequate, it may not be necessary to proceed further. Nevertheless, if the family history indicates a marked disposition to tuberculosis it may be wise to proceed further. In persons not so favourably situated, for instance in the poorer classes living in small, badly built, and badly ventilated rooms, and having scarcely more, and sometimes less, than the so-called necessities of life, in fact living under conditions which may lower tone and vitality, one should try and reach a high degree of immunisation by administering the more powerful varieties of tuberculin in large doses.

Old Tuberculin is one of the most potent forms, and is the cheapest of all tuberculins. Accordingly, in all the cases one meets at tuberculin dispensaries it is well to continue

treatment until 1, 2, or more cc. of Old Tuberculin have been given at a single dose. Maybe 1 cc. is sufficient for the majority of cases, but this remains to be proved. If after such a course of treatment relapse of the disease occurs, one may either repeat the previous course or use the tuberculin emulsion (T.E.).

Again, one may use either the bovine T.E. (P.T.E.) or the human (T.E.). This T.E. is given similarly in progressive doses until 2 cc., 3 cc., or 4 cc. are given at a single dose. It is best to give the larger doses in the back under the angle of the scapula or between this and the axilla; here the subcutaneous tissues are loose, and room can be found for the mass of fluid. One may inject the whole into one spot or distribute the amount in several places. Some prefer to use T.E. throughout for treatment, and excellent results are often obtained, but T.E. is absorbed with some difficulty, and causes much more discomfort after injection.

T.R. is another very valuable preparation, and works extremely satisfactorily in early and fever-free cases; but in my experience it does not work so well in advanced cases, and it is much more apt to cause severe reaction than the varieties P.T. and T.A.

Some authorities—Moeller, Friedberger—have used tuberculin made from the avian strain and also from the saprophytic form of tubercle bacilli; however, one sees no reason to either use or recommend these preparations.

General Remarks upon the Uses of Tuberculin.

While it is true that every medical man, even the general practitioner in the country, may learn to use tuberculin in all suitable forms of tuberculosis, it must be understood at the outset that it needs time, patience, and some experience in methods of administration, and especially in the system of progressive dosage, if uniformly satisfactory results are to be obtained. In the first place, the exceedingly high value of tuberculin as a diagnostic agent must be thoroughly realised and constantly exploited in all suspicious or doubtful cases. Invaluable experience, often with incalculable benefit to the

patient, may be gained by using tuberculin as the indispensable means of making an early diagnosis. It is in "contact" cases—especially that tuberculin alone can distinguish between infected and non-infected persons. This routine use of tuberculin at our tuberculin dispensaries gives an advantage to our system which belongs to no other system. At the so-called anti-tuberculosis or tuberculosis dispensaries, where tuberculin is not used in this absolutely routine way, mistakes are inevitable, and it is equally culpable to overlook a case already infected as to discover tuberculosis where it does not exist.

This was the great secret divulged to the world of science and the medical profession in Professor Koch's original communication in 1890. More than twenty years have passed, and still there are leading men in the medical profession who not only have rejected this great idea without any satisfactory and thorough investigation of their own, but even dare to question the truth of this magnificent discovery on evidence that would be rejected in any court of law. Not only does this method of diagnosis help us where all other methods fail, but it forms the unerring guide to diagnosis that leads to and culminates in successful treatment by specific remedies.

Of course, if tubercle bacilli are found in the sputum the tuberculin test is superfluous, but if we wait to establish the diagnosis in this way before we adopt specific treatment, we are losing the best opportunity of successfully curing the disease. There are many forms of tuberculosis in various parts of the body: in the glands, in the joints, in the bones, in the pleura, in the peritoneum, in the genito-urinary tract and elsewhere, in which the diagnosis of tuberculosis in no way depends upon the discovery of tubercle bacilli; and it is equally true that the diagnosis of pulmonary tuberculosis can be made with certainty even before tubercle bacilli have escaped from disintegrated tissue and reached the air passages. I may say with confidence that it has rarely, if ever, been my lot to discover tubercle bacilli in the sputum when there were not also to be found on careful examination obvious physical signs of the disease.

It must be clear to all who have a proper conception of the tuberculous process in the lungs, that for months and even years before tubercle bacilli escape into the air passages the tuberculous disease exists and progresses. Surely then, by waiting for the presence of tubercle bacilli in the sputum, we are deliberately closing our eyes to the supreme importance and real meaning of the early diagnosis of pulmonary tuberculosis, and granting the tubercle bacillus an undesirable and fateful opportunity.

There can be no progress in the scientific treatment of pulmonary tuberculosis unless the full and transparent truth of Koch's discovery wholly possesses our minds. If there still be medical men who reject this teaching, one can only hope that the laity themselves will become alive to the importance of this discovery and benefit by it. Until this truth is thoroughly recognised both by the profession and the laity there must be hundreds and thousands of lives sacrificed to scepticism, prejudice, and perverse opinions. The very highest authorities tell us that sanatoria fail because the diagnosis is not always made when the disease is in the early stage, and yet till recently very early diagnosis by means of tuberculin has been regarded as a mere fad or fancy; else how is it that in some of the London hospitals and even in sanatoria the practice of using tuberculin for diagnosis has not been universal? I can speak from experience, because for years¹ I was the only member of a large hospital staff who systematically in every case of suspected or doubtful tuberculosis never hesitated for a moment to use this diagnostic agent; and let me add, as a result I have frequently shown that there was no tuberculosis when this disease had been definitely diagnosed by others; and on the other hand I have proved tuberculosis to exist when others scouted the idea of any tuberculous disease. In two ways, then, tuberculin helps us in diagnosis, and two distinct forms of error can be eliminated in no other way. In a previous chapter I have more fully considered this problem in diagnosis, and I merely insist again upon its importance because unless tuberculin is used as the key to early diagnosis we

¹ Every member of the staff now uses tuberculin for diagnosis.

cannot achieve the high standard of success which should be the aim of every physician true to his calling and faithful to his trust.

I am ready to admit that some of the results observed after the use of tuberculin by those who either do not understand the dosage or fail to heed the express conditions and limitations urged by Professor Koch in the use of this remedy gave a semblance of justification to the view that the remedy itself was dangerous, and its effects capricious and even harmful. Even now there are those whose minds are obsessed with the idea that dangers, which can neither be anticipated nor eliminated, lurk behind every dose. It is strange, then, that in my experience, although I have used the very largest doses in every stage of the disease, I have not once seen a dangerous effect which I could trace to the remedy alone. Dangers there are, many of them, inherent in pulmonary tuberculosis and other forms of tuberculosis; dangers of generalisation in gland tuberculosis, in bone tuberculosis, in joint tuberculosis; in fact, in tuberculosis in any situation. But I am perfectly convinced that such generalisation after small doses of tuberculin, if it ever occurs, occurs with infinitely less frequency than after many surgical procedures. It is somewhat strange that the risk of generalisation in surgical procedures, a risk which has an obvious explanation, receives but scant attention, while the risk of generalisation after doses of tuberculin properly administered, a risk so small that I cannot admit its existence, has been grossly and unreasonably exaggerated. In my judgment, not only does the risk of generalisation after proper doses of tuberculin not arise, but the proper and judicious use of tuberculin clearly and positively diminishes risks that are a recognised feature of tuberculous disease in any organ.

Latent pulmonary tuberculosis producing no characteristic symptoms and exciting no suspicion in the affected individual, may be suddenly complicated with tuberculous meningitis. On the other hand, it must strike everyone who has studied the disease how uncommon it is for advanced pulmonary tuberculosis, even in the third stage, to end fatally by the supervention of tuberculous meningitis. Again, hemorrhage

is a very common symptom in pulmonary tuberculosis, and yet how capricious and uncertain. In my experience, in very many cases of pulmonary tuberculosis, complicated with hemorrhages of every degree, all the evidence goes to show that tuberculin is the best remedy for arresting hemorrhage and preventing its recurrence. Further, during the whole of my experience in Australia, in treating pulmonary tuberculosis in every stage with very large doses of tuberculin, I have not to record one single accident in which tuberculous meningitis supervened.

Finally, I became so supremely confident that even large doses of tuberculin in any stage of tuberculosis, though producing well-marked and severe reactions, did not thereby increase these risks that it had been my routine practice for a great number of years to treat many cases in all stages of the disease at a distance; sometimes without seeing the patients for months, and many months, and even years; and in no single instance had I any reason to regret that I carried out the treatment under these unusual conditions. These unusual conditions were forced upon me because many of the patients lived in distant colonies, and I took the risks in order to spare my patients the inconvenience and great expense that must otherwise have been incidental to the treatment, and in some cases must have put tuberculin treatment beyond the means and reach of the patient. It was not possible for me to ask a medical man to assist me in carrying out a system of treatment of which he did not approve. Thus by my boldness I both benefited the patient and further proved the innocuousness of this system of treatment if carried out with care and discrimination. I admit that, bearing in mind the views entertained by some concerning the dangers wrongly associated with tuberculin treatment, one must be bold who dares to treat pulmonary tuberculosis in this way, even at a distance of many thousand miles. I imagine that few men have had an experience of this kind, but as the experience has been entirely free from the smallest accident, it may be advanced as the strongest possible evidence of the truth that in proper hands there is no risk at all.¹ If

¹ I have since treated patients in India, Ceylon, and South Africa from London.

accidents have happened in the experience of others, either the accidents were the effect not of the tuberculin treatment, but of the disease itself; or the remedy must have been used in a way different from my use of it.

In London also I have been using the remedy at the tuberculin dispensary indiscriminately in all forms of tuberculosis, in which I was certain that no "mixed infection" was present; and in this prolonged experience also I have not had one single accident. I am glad also to be able to say that many of my own pupils treating, some of them, a large number of cases, some eighty, some sixty, and others in smaller numbers, have had much the same experience. One of my pupils had a very narrow escape. Dr. Hilda Clark had decided to use tuberculin, but postponed the dose for no special reason. Within the time this patient died suddenly of a complication. If by chance that dose had been given, the fatal issue would have given a great shock to my newly found disciple, and might have seriously jeopardised the success of the method in her own district. Another pupil of mine relates a similar experience.¹

Generalisation of tuberculosis is no uncommon accident, and if perchance tuberculin is exhibited shortly before or after such generalisation has occurred, the man of limited experience may readily and wrongly come to the conclusion that the generalisation has been determined by the use of tuberculin. It is to be expected that some men will give doses of tuberculin when generalisation or other accident is imminent, and it is inevitable that such an one may be too timid to deal logically with the unfortunate, but purely accidental, situation.

It has occurred to me once to be brought face to face with such a situation, and I am honest enough to admit it. A patient consulted me for general failure of health due to obvious pulmonary tuberculosis, which had failed to be benefited by prolonged sanatorium and open-air treatment. I advised tuberculin treatment. The patient went away, and did not submit to treatment. Some months later he came to see me again. I observed that he was distinctly

¹ "Studies in Pulmonary Tuberculosis," P. G. Griffiths, M.D. 59d.

worse. He had headache and some insomnia. He was taciturn and depressed, but still these symptoms did not suggest to me any serious brain trouble. I hesitated to treat him, but finally yielded. I gave him one very small dose of the weakest preparation I use. This dose raised the temperature a little above 99°. There was no other effect, and no marked reaction at the site of injection, showing that the dose I had given him had hardly had any effect; yet within a few days this patient had obvious signs of tuberculous meningitis, to which he succumbed. As a matter of fact, the symptoms of meningitis even in this case occurred too soon after the injection to have been casually related to it. On further investigating the case, it was clear that he had had symptoms somewhat suggestive of cerebral irritation, and even his own doctor admitted this, and was convinced that the dose had nothing to do with the sad result. The mistake I made was in beginning treatment three months after I had carefully examined him without making another careful examination. During those three months his marked deterioration of health and increase of brain symptoms, which brought him back to me again, were no doubt due to actual tuberculous disease of the meninges. Possibly, if he had had treatment when he first came to me he might still be alive. The time had passed at the second visit, and it was a mere coincidence that I had given him an extremely small dose that could have had little or no effect. I tell this history because it is the first accident in my experience in which tuberculous meningitis has ever shown its presence by symptoms shortly after a single small dose of tuberculin. It would be mere foolishness to suggest that the relation was not *post* but *propter hoc*.

Although in my own practice I have used tuberculin for the most part in the treatment of pulmonary tuberculosis, I have used this remedy also in practically all other forms of tuberculosis with equally successful results. I am not able to speak of any experience in treating lupus, because lupus is almost an unknown form of tuberculosis in Sydney and New South Wales, but in pleurisy, joint and bone tubercu-

bosis, tuberculous peritonitis and tuberculosis in any organ, specific treatment may be carried out on similar lines and with equally satisfactory results. Certainly in early forms of joint and gland and bone tuberculosis hyper-sensitiveness is very common and may require patience and perseverance for weeks and months before the over-sensitiveness is mastered. Then there is no trouble in continuing the treatment until large doses can be given.

I have treated several cases of gland tuberculosis in which already extensive operations had been performed by surgeons without any remarkable success, and the impression has been left upon my mind that after these surgical procedures the results are not so satisfactory. It is my firm belief that in the future, when the value of tuberculin in diagnosis is universally recognised, by the exhibition of tuberculin for treatment in the very early stages of the disease, there will be less and less need for surgical operations. It is quite wrong to speak of these forms of tuberculosis (in joints, in glands, in bones, etc.) as forms of surgical tuberculosis, because early treatment by specific means will prevent those unfortunate catastrophes, such as enlargement and softening of the glands after extensive caseation, which have necessitated the use of the surgeon's knife. In the future all these forms of tuberculosis will come back into the hands of the physician who, with his increased knowledge and improved methods of treatment, will be able to effectually cure these conditions without any operations. Once the disease has gone so far that operation has become necessary, the prospect of ultimate and complete recovery becomes more and more distant.

Before leaving this subject, I feel it my duty to insist upon the fact, the importance of which is not sufficiently recognised, that the early diagnosis of tuberculosis in any form is often difficult. In the first place, the unfortunate victim may not suspect the real nature of the trouble till the disease is well advanced. Accordingly, it is not the doctor so much as the patient who is at fault in postponing the visit to the physician until the disease is well advanced. But I fear that too often the physician has the opportunity of making an

early diagnosis, but fails to seize it. I have known many instances in which laryngologists have been consulted for coughs and hoarseness, and forgetting that they cannot see beneath the surface, they give the name of catarrh or hysterical aphonia to conditions that were the early signs of tuberculosis. In like manner the off-hand diagnosis of dyspepsia or anaemia or hysteria or debility or rheumatism or even asthma or bronchitis is made, when the real cause of these states of ill-health has been overlooked. I have had illustrations of all these mistakes in diagnosis in the hands of medical men who eschew the use of tuberculin for diagnostic purposes; for, not long afterwards, sometimes days, sometimes weeks, sometimes months, these patients have come to me often with obvious signs of the disease in their lungs or elsewhere, all reacting in the very profoundest way to tuberculin and responding so well to tuberculin treatment that they lost their symptoms and recovered their normal health. Not only specialists in the larynx, in diseases of the nervous system, in diseases of the stomach, in diseases of the blood, in diseases peculiar to women, but, in fact, in almost every field of medicine the physician must be on the alert and ready to use tuberculin in diagnosis, or he may allow his patient to be inflicted with an injury which can never be undone.

But it is also for the general practitioner, and above all for the family physician, to constantly bear in mind that one of the commonest causes of vague and variable phases and forms of ill-health may be due to this disease, which delights to masquerade under many guises of ill-health with indefinite characters that too often baffle our powers of diagnosis. I have even to confess that patients have come to me with evident signs of pulmonary tuberculosis, and yet very shortly before some practising physician had snipped off their uvula for an irritating cough. The family physician, who plays such an extremely useful part in social life, can greatly increase his usefulness by realising that tuberculosis is a very common disease and may be easily missed, sometimes with irreparable harm to the patient who trusts him, if the early diagnosis is not made by the

only method which leads us to a sound and infallible opinion. I am conscious of having prevented innumerable tragedies by adopting this plan, and I cannot urge too strongly that this practice should be universally adopted by all physicians, for they can have no better way of rendering a faithful and beneficent service to those who so implicitly trust them. I do think that the family physician has more opportunities of checking the ravages of tuberculosis in the community than any other class in the medical profession, because tuberculosis is essentially a family disease, and the family physician is so continually concerned and interested in the family life that he has many opportunities of proving his skill in diagnosis and treatment. If we can once win the family physician to the view that early diagnosis is the secret of success in treatment, not of course by mere statements, but by personal conviction, we shall have made the greatest step forward in the conquest of this disease. It is by means of the tuberculin dispensaries scattered throughout towns and in the country that we shall be able best to carry home conviction to the family physician of the great truths that have their origin in the life-work of Professor Koch. Each tuberculin dispensary becomes a centre of influence and education which must have a profound effect upon the whole question of the treatment of tuberculosis by specific methods.

Lastly, specific treatment by means of tuberculin opens up a magnificent vista of usefulness to female physicians, to whom many avenues of work are likely to remain closed.

PART VII

THE RÔLE OF THE TUBERCULIN DISPENSARY
IN THE CRUSADE AGAINST CONSUMPTION

THE TUBERCULIN DISPENSARY

PART VII

THE TUBERCULIN DISPENSARY

THE problem of dealing with pulmonary tuberculosis, and indeed other forms of tuberculosis among the poor by means of tuberculin dispensaries, is, so far as I know, quite a new phase of an old problem. I do not know of any published records, apart from my own, in which an attempt has been made to prove that in cases of pure tuberculosis specific treatment alone achieves results that cannot be approached by any other system. In order to test this question in a logical way one must avoid the introduction and prevent the intrusion, as far as possible, of factors which in themselves may contribute something to the successful result. There are those who consider that abundance of fresh air may make all the difference between success or failure in the treatment of pulmonary tuberculosis. Thus, if we wish to prove that specific remedies are the effective agents in curing the disease, we must allow the individual to continue to live under the conditions to which he has been accustomed. Again, some think that over-feeding may work miracles in curing tuberculosis. Therefore, in testing the value of tuberculin one must order no change at all in the diet of the individual. Then if the specific remedy is the one factor deliberately isolated and exploited for treatment, any successful issue can be due to nothing else but this single and antecedent factor. I do not know of any authority except myself who has attacked this great problem in this particular way.

In Germany and elsewhere, as in America, it is a common practice to use tuberculin, too often to play with tuberculin, in sanatoria. Under such conditions it is hopeless to expect to trace the effect to its proper cause. Thus the sanatorium

system is at work, and in some way or other tuberculin may be doing something. But who can say how far each of these factors is contributing to the result? Those authorities who have merely used tuberculin in sanatoria may easily attribute results due to tuberculin to the well-known régime of the sanatorium. It is, of course, obvious that the more stimulating and agreeable the environment, the better able are tissue cells to resist deleterious agencies of any sort; yet, in spite of the best environment it is well known that pulmonary tuberculosis may begin, progress, and terminate in death.

Of recent years the results of sanatorium treatment have been better than in former times, and in many instances this better record is the direct result of the increased use of tuberculin in sanatoria. But in dealing with the problem as it affects the poor, it is of the highest importance to differentiate between the effects produced by the so-called sanatorium methods alone and those caused by tuberculin alone, so that we may compare them and arrive at a sound conclusion upon the relative merits of the two systems. If it can be shown that the simple, direct method of specific treatment yields results even equal to those of sanatorium methods, there is no doubt that sanatoria must give place to tuberculin dispensaries in the solution of the problem as it affects the poor. I am convinced by my own records, in which I have carefully avoided the use of sanatorium methods and other methods that involved loss of occupation and wage, because I have carefully studied what specific treatment *per se* has been able to achieve. I therefore trust that all those who may differ from me on this question will at least remember that I am merely adducing evidence in favour of my contention that, if for poor people tuberculin treatment be as effective as I have shown it to be in my experience—far more effective, in fact, than sanatorium treatment—it is the obvious duty of administrative bodies to substitute a method which is simple, economical, and effective for a method which is largely inapplicable, wholly inadequate, and for the most part ineffective. I admit that sanatorium treatment is of some value if time and money and work are of no account, but for the poor time, money, and work are

of great account. Even rich people who have everything within their reach, and may, if they choose, spend all their days in sanatoria, too often find to their disappointment and sorrow that ill-health still dogs their footsteps. At most the poor can find entrance once into a sanatorium, and then must return to their home and home conditions. How often they return home cured, no sanatorium authority dares to say. Is it not inevitable that sooner or later the disease will return? Then what is their condition? Can sanatoria help them any more? How different is the purpose, method, and scope of the tuberculin dispensary! Let us consider its purpose first.

The primary and essential function of the tuberculin dispensary is to offer to the poor afflicted with consumption a method of treatment which holds out a good prospect of cure. So poor is the prospect of cure for the poor by treatment in sanatoria, that nowadays the word "cure" is expunged from sanatorium literature. On the other hand, at the tuberculin dispensary it is possible, as Professor Koch has himself claimed, and his pupils have reiterated the claim, that in the early stages pulmonary tuberculosis can be cured with certainty by tuberculin. No doubt in the early stages the disease may sometimes cure itself, and too many such cases go to swell the vaunted successes of sanatorium treatment. Moreover, in the records of many institutions one fears that the diagnosis has been too early. I distrust altogether any records dealing with early cases of pulmonary tuberculosis unless the diagnosis has been verified by means of the tuberculin test. In my own experience I had to repudiate the diagnosis of pulmonary tuberculosis made by recognised authorities, in perfectly good faith, but without the scientific test by means of tuberculin, in no less than sixty cases. On the other hand, I proved tuberculosis to exist where the idea of disease had been scouted in about an equal number of cases. Of course, if one treats healthy cases as cases of tuberculosis, and rejects cases of actual tuberculosis, one is likely to obtain successful results with any methods of treatment.

One learns that the Charity Organisation Society is

carrying out investigations into the subsequent history of patients who have been treated in sanatoria, by aid of their medical advisory committee. The whole value of this work depends upon the methods used by this medical Committee in arriving at correct diagnosis in the first instance, and the character of the after-examination. It seems remarkable that the Charity Organisation in the course of eight years should only report the after-histories of less than 500 cases. It is obvious that they have reported but a limited number of those that came under their observation, and this alone renders such an examination almost worthless. What is the use of such figures when we have the figures of Weicker and Engelmann and others, dealing with 30,000 cases, in which the results were very different from those collected by the Charity Organisation Society? For all we know, 200 of these cases may have only a history of a year or two behind them.

The primary object, then, of the tuberculin dispensary is treatment. We thus appeal to the strongest motive in human nature—self-interest, and if the treatment be successful we shall not want for lack of patients. Other systems appeal rather to the intelligence of the people, and even deliberately avoid treatment lest they should interfere with the work of the medical practitioner. I should be loth to encroach upon the practice and work of anyone who offered a satisfactory method of treatment to his patient, but at the present moment there are very, very few practitioners who know even the rudiments of successful treatment by tuberculin.

As I have said already, the tuberculin dispensary is the place for the medical practitioner to study and learn this scientific method of treatment. Then the officer at the tuberculin dispensary will be glad to be relieved of some of his patients, but meanwhile the interests of the patient are the first to be considered. I am able to place on record the simple fact that the medical men in the district around the tuberculin dispensary have gladly sent their poor patients to the dispensary, and have themselves visited the dispensary to learn the methods of treatment. This is as it should be, and

I trust that on these principles we may avoid any friction or unpleasant collision with the medical men. I hope that at the chief dispensaries, as at the large hospitals and special hospitals, some of the work may be undertaken and carried on voluntarily and gratuitously.

Tuberculin dispensaries exist only for the relief of the poor, and if medical men attending upon the poor are competent to carry out this highly technical method, the reason for tuberculin dispensaries disappears, but we are still far off the millennium. Meanwhile, the medical men can hardly feel aggrieved if this simple and beneficial treatment is placed within reach of the deserving poor. At the present time, too, the attitude of the medical profession is so sceptical that these institutions are necessary to prove whether their scepticism is at all justified. Every day the advocates of tuberculin treatment are growing in number, but hitherto no system of tuberculin treatment *per se* has been given a serious and thorough trial in Great Britain. It will, I trust, be the special aim and purpose of the tuberculin dispensaries to settle beyond all cavil or doubt whether tuberculin used in a definite way in a large series of cases can achieve successful results. By that I mean arrest of the disease not merely for a few months or a year or two, but for at least four or five years, and perhaps for many years longer. If we succeed in showing that, in the majority of cases, classified according to the stages familiar to us, better results are obtained by the tuberculin dispensary than by other systems, there will be an enormous gain to the community, because tuberculin treatment is far less costly than sanatorium treatment, and may not involve any sacrifice of occupation or earnings.

This problem of proving what tuberculin alone can do in the treatment of tuberculosis is of such supreme importance that no pains should be spared in determining once and for all time this question. It is not enough merely to pay tuberculin back-handed compliments by saying that it is valuable in specially selected cases (Philip). This tells us nothing. Many things may be valuable, such as drugs, aperients, cough mixtures, and the like. What we want to

know is whether tuberculin is a specific remedy for all forms of tuberculosis. If so, we wish to know also which is the best form and in what manner this remedy should be administered. I admit that the remedy has such a striking and direct effect upon the tuberculous process that even in small doses it may cause very great improvement, with lessening of all the striking symptoms, such as cough, expectoration, loss of energy, failure of appetite, sweating, etc., but such small doses have not the power to eradicate the disease. Accordingly, in order to give a satisfactory solution to an investigation in this direction, we must determine at the outset what is the object of treatment. If it be merely to alleviate symptoms and cause temporary improvements, such small doses may be adequate, but if the object of treatment is not merely to allay symptoms, but to attack and destroy the actual living cause of the disease we must use the valuable remedy boldly and without stint until the tissues are rid of or beyond the reach of the living bacillus.

No doubt the methods by which we try to prove the complete efficacy of the remedy may not be absolutely conclusive or entirely satisfactory. Still, there are methods that may help us, such as Wassermann's method by fixation of the complement, and Arnet's method by a differential count of the polymuclear neutrophil leucocytes, or perhaps even the opsonic index, although this last method is not so trustworthy as the first two.

It goes without saying, too, that in such an investigation the influence of climate and accessory methods, such as those of the sanatorium, must be eliminated. At least while it is held that the value of tuberculin in the treatment of tuberculosis is still *sub judice*, it is right to insist that the investigation should be made so as to exclude factors which may have a disturbing influence. Naturally sanatorium authorities desire that tuberculin treatment should be carried out in their institutions, because then, with a semblance of truth, they may claim for their system the advantages which are due to tuberculin. Many sanatorium authorities have exploited tuberculin in cases which resisted sanatorium methods, and were surprised to find that

tuberculin treatment often succeeded in a striking way when sanatorium methods had egregiously failed. If they had but prosecuted their experiments with tuberculin in the right way, they would have found that tuberculin also succeeds where sanatorium methods succeed, and the success with tuberculin reaches a far higher level, because the results last.

It is remarkable that no serious and continuous efforts had been made to differentiate between the effects due to tuberculin alone and those due to sanatorium methods, until I set myself to this task. It is well known that for very many years I have repudiated all the methods in vogue in sanatoria and allowed the patient under treatment to live under exactly the same conditions during treatment as before treatment, except that tuberculin has been given. I have therefore been able to prove that tuberculin alone used in progressive doses, increasing them until very large doses are given, not only rapidly relieves the symptoms, but restores the patient to a condition of sound health. This normal health, too, is maintained in a striking way for many years after the specific remedy has been suspended. Not only in early cases, but even in moderately advanced and well advanced stages of consumption, these extraordinary improvements and restoration to health are more frequent and more enduring than can be obtained by the best sanatorium methods if tuberculin is excluded. Of course, if tuberculin is used as I use it, in a sanatorium, the sanatorium will not interfere with the action of the tuberculin.

My contention, however, is that in a large proportion of cases, perhaps in all cases of pure tuberculosis, tuberculin alone is the sufficient and efficient remedy. Surely, then, we should place within reach of the poor institutions where this method of treatment may be obtained, especially if experience proves, as I stoutly maintain, that the treatment can be carried out, not only in the early stages, but even in the second stage of the disease, while the patient still remains at his work and continues to be the mainstay of his family. As soon as treatment encroaches upon the means of

livelihood of those who are just able to make ends meet, just able to keep the home together, and feed and clothe the members of it, we begin to see the slow disintegration of the family unit. By offering treatment which does not thus encroach, we save the family. And after all, the integrity and stability of the family unit is the very basis of our social system.

If it be true that sanatoria cannot help the poor who suffer from tuberculosis, and that tuberculin is an efficient remedy for the disease, it is clear that institutions should be established without further loss of time where the poor can be treated with tuberculin. I seriously proposed in the year 1897 that such institutions should be established instead of building sanatoria, but no one, either doctor or layman, would listen to me. A few years later I tried to carry out my idea in another way. In order to force the problem upon the attention of the municipal authorities in Sydney, I became a candidate in one of the municipal wards and was elected alderman. I brought my scheme before the Municipal Council of Sydney, and it was adopted unanimously, but through some legal difficulty my plan was stale-mated. I have never wavered in the idea that dispensaries were the simplest, cheapest, and best means for bringing this special treatment for consumption within reach of the poor.

These dispensaries must be special and independent institutions, devoted exclusively to the treatment of tuberculosis of all forms by means of tuberculin. It is a mistake to imagine that tuberculin treatment might form a part or branch of the work of the ordinary dispensary. Tuberculosis is so common that a dispensary for the tuberculin treatment of tuberculosis has far too much to do in treating the cases of tuberculosis to give any of its time to treating any other disease. Besides, the treatment itself needs so much care in detail and exacts such undivided attention that it would be unwise to associate this work with any other medical work at a dispensary. Everything too, all the injections, must be given with the usual aseptic precautions. The graduation of the doses, too, is so utterly unlike any other system of treatment that serious risks may

be run unless the system of giving to different patients doses of very different strengths is carefully and minutely watched and controlled so that wrong doses are never given. The several patients are in different stages of treatment, and at a certain stage a dose may be given without the slightest effect which, if given to another case in a different stage, might have disastrous results. I do not think it likely that anyone who has had any practical experience at such a dispensary could conscientiously disagree with me. I quite allow that if we were merely using the system of minute doses without progressive increases that this objection would not hold, because it does not matter how you mix up these minute doses in different or in the same persons, but with the system of progressive doses, which are ultimately measured by cubic centimetres, the situation is entirely different.

I think it will always be necessary to continue these tuberculin dispensaries as independent institutions, though of course any hospital may possess such an independent institution. These tuberculin dispensaries can be called by no other name, because they are dispensaries at which the essential remedy for treatment is tuberculin. They differ in this respect from the anti-tuberculous dispensary, which is hardly a dispensary at all, but is rather a place or station where patients are examined so that some may be sent to homes for the dying and some to sanatoria, while a few may be treated by drugs.

The Edinburgh system, which Dr. Philip advocates, aims at prevention, and has hitherto treated the cases essentially on the old lines. At any rate, I know that when I started my tuberculin dispensary in London Dr. Philip did not approve of the use of tuberculin, either for diagnosis or treatment at his dispensary. I am inclined to think that my work in London has compelled him to modify his system. He still clings to sanatoria, and that in itself furnishes the clearest proof that he has not exploited tuberculin with any great success.

The primary and essential purpose of the tuberculin dispensary is to treat all forms of tuberculosis among the poor.

and this is the special business of the physician. No doubt the physician may also help the cause and work of prophylaxis by treatment. Thus every case cured by tuberculin is one less source of infection; but the main business of prevention must be left in the hands of the Medical Officers of health, else there will be wasteful and awkward overlapping of duties. I am sure that it is the best economy in this work to recognise the principle of division of labour, and therefore I trust that the whole energy and time of the physician in charge of the tuberculin dispensary shall be devoted to the *treatment* of tuberculosis. There are all too many sufferers sadly in want of treatment, and as soon as their wants are fully satisfied, the physician may think of devoting his attention to prevention.

A modification of the Edinburgh system has been introduced at Oxford, chiefly through the influence and advocacy of the Regius Professor of Medicine, Sir William Osler. This system is tantamount to an admission that sanatoria have failed as a means of treating consumption among the poor, since the advocates of this system propose that the patients shall be sent to a sanatorium for a month or so, in order to have the principles of personal and domestic hygiene instilled into their minds. This may be a picturesque way of gently breaking off one's allegiance to an old love. There is some justification in this attitude, when one has exhausted one's *Schwärmerei* for the old idea, and has not yet had the courage to embrace the new idea. Meanwhile, the old idea is not to be faithlessly discarded, but may be for a brief space exploited in another way—for purposes of education. One needs to have much faith in the intelligence of the needy victims of consumption to suppose that a month's residence in a sanatorium can powerfully modify the ingrained and tenacious habits of heredity strengthened by the environment of a lifetime.

If this change in the mode of life can be brought about at all, the change must be gradually and steadily made in the home itself. Accordingly, if sanatoria have failed as a means of treating the poor who suffer from consumption, it is far better to spend eight or ten pounds in im-

proving the home conditions and in instructing the poor in their *own homes* in matters of personal and domestic hygiene than in support of sanatoria. The mother is the mistress of the home, and it must be through the mother that the changes in the family-life are introduced. The invaluable and indispensable agent in this work of levelling up the standard of hygiene in the homes of the poor is the properly trained district nurse who instinctively understands the difficulties and limitations of the housewife, and is able gradually by iteration and reiteration to improve the home conditions of the poor. I do not think that the young doctor by an occasional visit can bring about much improvement that will last. Certainly, nowadays, the fully equipped lady doctor may be able to do this kind of work even better than the district nurse. Looking into the future, I fancy I see women, who are also doctors, playing a far more important part in the treatment and prevention of tuberculosis than in any other branch of medicine, except in the special treatment of women. When this phase of natural selection has had time to develop this passing idea of Sir W. Osler's, which is, after all, only a weak compromise, of attempting to instruct the poor in sanatoria will hardly have an historic interest.

If, indeed, a month's instruction in sanatoria is necessary, we should not confine this valuable instruction to the poor, but we should give similar instruction to many who, though not poor, offend quite as often and as grossly against the laws of personal and domestic hygiene. All this may be salutary enough, but it hardly touches the serious problem before us of *treating* consumption, and it is assumed that the poor will be ready to acquiesce in these pleasant excursions to sanatoria for their schooling. Such lessons would be of the greatest value to those who are in the later and latest stages of the disease, but I imagine that these are the very cases that the sanatorium authorities will be reluctant to admit. There may be some reason for sending early cases to sanatoria for treatment, but it is recognised that less than six months' treatment has not much permanent effect upon these early cases. A month's stay at a sanatorium could

have no effect upon the disease itself, but the money spent in this way, if devoted to tuberculin treatment at a dispensary, would be of enormous benefit to the patient. I cannot imagine by what influence the poor would be persuaded to make sacrifices in order to spend a month in a sanatorium if they thoroughly realised that this disciplinary period of a month was not to benefit themselves, but for the uncertain, indefinite and doubtful benefit of others.

In all I have said, I wish again to lay stress upon the simple fact that I am merely criticising this system of disciplinary instruction in so far as it applies to the poor. I wonder how many of the victims of consumption in the better classes of society would submit to such a travesty of treatment, and I should not wonder if the poor, as soon as they understood the purpose of the scheme, resented and rejected it. Some might, indeed, look upon it as a sort of holiday trip, and take advantage of this somewhat officious form of charity, but I should expect the more intelligent to refuse to sacrifice their positions and their earnings, even for a month, for such an intangible object. It will probably be borne out by experience that those who would most benefit by this system would be the last to offer themselves as pupils.

In carrying out a system of this kind, too, we may be actually doing harm by sowing the seeds of discontent in those who have to return to their own wretched homes after a month's stay in a sanatorium. It is easy to imagine also that a man who has been taken away from his work may be very reluctant to begin work again when he has weakened the habit of work by this holiday. For this reason one can applaud the system of graduated labour or graduated exercise. The pioneer of this work was Behmer, who planned out the pine forests at Goerbersdorf so as to regulate accurately the amount of walking exercise allotted to each patient. A modification of this system is now practised at Frimley and elsewhere. The real pioneer of this work was Behmer, who, working on an entirely erroneous theory, evolved a sound, practical system. If hard labour was made part of this disciplinary régime some of

the objections to this system would be mitigated. I still hope that before this system is actually put into practice the popularity and usefulness of the tuberculin dispensaries will render this restricted term of instruction at a sanatorium quite superfluous.

Besides, sanatoria will play a useful part in this problem when they are devoted mainly to the treatment of cases of mixed infections and of other complications of pulmonary tuberculosis. It is far more important, and a much sounder policy, to devote sanatoria to these cases than to fill them with individuals who can be better treated and better instructed in their own homes.

In order to give uniformity and solidarity to this new and eminently practical way of treating the poor, who suffer from tuberculosis, at tuberculin dispensaries, the Tuberculin Dispensary League has been established, and I append a succinct statement of the purpose of this League, which was written for the *British Journal of Tuberculosis* by the Countess of Mayo, the President of the League, and myself. I wish also to acknowledge the splendid assistance the League has received from our enthusiastic and energetic secretary, Mrs. Stuart Erskine.

THE TUBERCULIN DISPENSARY LEAGUE

This new league justifies its existence, because it is exploiting a new method of dealing with tuberculosis, especially pulmonary tuberculosis among the poor, in a way never before attempted, and its work does not overlap, and is not overlapped by the work of any other society or system. The primary essential and distinctive purpose of the Tuberculin Dispensary League is to bring within reach of the friendless poor some simple and effective method of treatment.

Public Assistance for the Tuberculous Poor.

Can sanatoria greatly help the poor? is a question that can only be answered in the negative. If the friendless poor can be treated for tuberculosis in sanatoria, there is no need for tuberculin dispensaries. If they cannot, tuberculin dispensaries become an urgent necessity. A cursory consideration of the problem will prove why sanatoria cannot greatly help the poor who suffer from tuberculosis.

Tuberculosis kills at least 55,000 persons every year in England and Wales, and there are always at least four times as many persons—220,000—victims of tuberculosis who need treatment; and of these the vast majority are too poor to have treatment at sanatoria. Even if sanatoria could treat half of the cases, is nothing to be done for the remainder?

The cruel fact is that sanatoria can only treat about 20,000 cases out of this 220,000, so that 200,000 are left without proper treatment. The treatment of 20,000 cases at sanatoria cannot cost less than £600,000 a year; the cost of treating all the cases at sanatoria would be about £6,000,000 a year. Now, if sanatoria, stretched to their uttermost, can only deal with 20,000 cases, it is high time to look about for some simpler means of relieving this huge contingent of 200,000. Beyond a doubt the tuberculin dispensary is at present the only means of granting this relief; and for many, many years tuberculin dispensaries will have far more than they can do without interfering in the least with existing sanatoria. Even if the work of sanatoria were doubled and

trebled, the majority of sufferers must seek relief from some other system or die. In truth, there are sanatoria enough without adding others, because the sanatorium system is too irksome, too costly, and too uncertain in its results to benefit the poor in their extremity; the sacrifices are too great and the successes too few. The benefits of sanatorium treatment do not last very long, if the victims have to return to their ordinary avocations and conditions of life; and life at a sanatorium may even demoralise a man and engender the seeds of discontent. The inevitable loss of work and wage alone disqualifies the sanatorium as a reasonable system for those who have to live upon the daily wage, except indeed under the German system. Still, one may tolerate and even applaud the provision of sanatorium treatment for 20,000 cases at a cost of £600,000 a year—certainly at the hands of charity, but not at the public expense—without in the same breath condemning 200,000 victims to remain without any satisfactory treatment.

The Role of Tuberculin Dispensaries.

Tuberculin Dispensaries are to be the means of giving simple, direct, cheap, and effective treatment to the huge majority inevitably neglected and rejected by sanatoria. Treatment at the dispensary is direct and specific, as distinct from the so-called rational or hygienic diietetic system practised at sanatoria. Specific treatment embraces the use of the various forms of tuberculin which have been presented as a free gift to a thankless generation by one of the world's greatest benefactors, Professor Robert Koch. At the tuberculin dispensary, also, tuberculin is exploited as the invaluable and indispensable agent in diagnosis in accordance with the views first enunciated by Koch in 1890, which have year by year attracted greater and greater attention and support. In fact, tuberculin is used in a routine fashion as the diagnostic agent in all suspicious or doubtful cases, especially among contacts who have been exposed to a serious risk of infection. For diagnosis both Koch's original method and Von Pirquet's method are used. Calmette's

ophthalmic reaction is discarded for the best of reasons: that if tuberculin be subsequently used for treatment, the conjunctival reaction repeats itself for some time in a most objectionable way after each subcutaneous therapeutic dose. The routine use of tuberculin in diagnosis greatly enhances the value of tuberculin as a remedy, because the tuberculosis can thus be detected in the very early stage, when no less an authority than Professor Koch has taught us that the disease can be cured with certainty by means of tuberculin. Other observers have corroborated this opinion of Professor Koch which appeared in the historic communication of 1890.

But the advocates of tuberculin are not so foolish as to base their advocacy of this remedy merely upon the results obtained in these early cases, even though there may be almost 100 per cent. of successes, because in this stage success often attends no treatment at all, and occurs very often after prolonged sanatorium treatment. It then becomes a question of apportioning successes and failures, and we may readily remember our successes and easily forget our failures, or leave others to record them. The value of tuberculin treatment is most clearly shown in the later stages of pulmonary tuberculosis, especially when severe laryngeal lesions, ulcers, and granulations have developed. It is then possible to watch directly the curative action of the remedy in cases that are otherwise quite hopeless. It is a matter of history that Moeller, of Belgig, and others had the truth of the value of tuberculin as a curative agent first forced upon their attention, when they tentatively used tuberculin in cases that had resisted sanatorium methods. Tuberculin brought about improvement that was as startling as it was unexpected. It was then suggested to them (Camac Wilkinson) that if tuberculin succeeded when sanatorium methods failed, tuberculin might also succeed when sanatorium methods succeeded. Moeller has convinced himself of this fact, and the quondam advocate of sanatoria is now the most outspoken advocate of tuberculin.

It is also certain that sanatoria may succeed where tuberculin fails, but it has been no easy matter to convince sanatorium authorities of the real nature of these cases and the

reason of the failure. Tuberculin is absolutely specific in its effects, and can influence favourably no other infection than tuberculosis. Unfortunately, pulmonary tuberculosis may be combined with infections of other origin, chiefly streptococcal, pneumococcal, and influenzal, when injections of tuberculin may do distinct harm. These mixed and secondary infections have the further objection that they increase the over-sensitiveness to tuberculin (anaphylaxis). As yet no specific treatment of these mixed infections has been found to yield any uniformly satisfactory results, and until such specific treatment of the mixed infections has been evolved, *sanatorium treatment is quite the best for these mixed infections*. Accordingly for the present, sanatoria should be specially devoted to the treatment of these mixed infections (Petruschky), while all forms of pure tuberculosis should be treated with tuberculin. Sad to say, sanatorium authorities cling to topsy-turvydom, and seek for the early cases which are best treated with tuberculin, while they reject the cases of mixed infections which may be enormously benefited by sanatorium methods.

A casual observer may argue that if this be true, a sanatorium is surely the best place both for the treatment of tuberculosis by tuberculin and the treatment of the mixed infections. This is a stock argument that appeals to the unthinking, and runs right through the facile writings of those who have no sense of proportion and forget the limitations of time and space and money on our poor little planet. If time and work mean nothing, and sanatorium treatment can be had for the asking, certainly the sanatorium is the place for all of us; but why not build "castles in the air," and fill them with patients? If all the hospitals in England were turned into sanatoria, we could not house all the cases of tuberculosis. No; any scheme for dealing with tuberculosis must recognise facts; and the facts stated in the earlier part of this article prove that sanatoria can never deal with the serious problem of tuberculosis in the community.

Practical Measures.

It only remains for us to show that tuberculin dispensaries offer a simple, safe, economical, and effective method of treating the disease in the poorer classes of society, and it must follow that tuberculin dispensaries will be established in every community as the best solution of this great national problem—at least at the present time. The proof lies, not in vain theorising, not in pandering to prejudice, but in experience. Already there are enough of these tuberculin dispensaries in existence for everyone to form an honest and independent opinion upon certain features. The system is very simple, and the popularity of the institutions alone is presumptive evidence that the system is safe and useful. Already many scores of patients have been treated with very great benefit. It is within the mark to say that for tuberculin treatment the cost need not exceed £2 for each case treated. It has also been shown that at least 80 per cent. of the cases can be treated without any sacrifice of wage or work. Time alone can show whether the results will last; but there is much evidence in favour of the view that the results will last longer than sanatorium results. Surely, then, there is reason and truth and wisdom in the statement that if the money now devoted to sanatoria for London were devoted to tuberculin dispensaries, not one thousand, but actually *twenty thousand*, or even *thirty thousand* sufferers could be treated each year, and the individual results would be better. Surely "a consummation devoutly to be wished."

Tuberculin dispensaries are simple institutions at which poor people can be treated for consumption by injections of tuberculin. They should, as far as possible, be worked upon the same plan, so that poor people migrating from one district to another may continue treatment without interruption. The treatment should be continued for at least six months, but this need not be very irksome to the patient if he is able to attend the dispensary at stated hours. For the poor no doubt the evening is the most convenient time, and I doubt not, that in the future, tuberculin dispensaries will be

open in the evening. Employers find it difficult to allow their employees to leave their work for an hour or so twice a week in the morning. It is even worse for the employee when the employer has the notion that those who attend at the dispensary are a source of danger to their companions or fellow-workers. It is surely better that the employer should encourage the employees to have effective treatment at the dispensary, which in early cases effectually prevents any risk of infection, rather than force the employee to neglect himself and avoid the dispensary until he has passed into the infectious stage of the disease. I am sure that it is wise for the employer to help rather than thwart their employees in this praiseworthy anxiety to recover health. As their health falls, the quality of their work deteriorates; as their health improves, the work they do for their masters is certainly of improved quality. In the employer's own interest, in the interest of the suffering employee, and in the interest of his fellow-workers, it is highly desirable that the disease should be detected early and treated systematically, and the tuberculin dispensary alone offers a simple solution of the patient's dilemma. At most, the employee has to sacrifice about three or four hours a week, and this sacrifice is worth making, and the employer should help his employee to make it. I have had two instances in which the patients suffering from evident disease had to give up treatment because their employers refused to allow them to visit the dispensary twice a week.

These dispensaries must be numerous in large towns, because it is not possible to treat a very large number of cases at one institution. I should hope that many of these tuberculin dispensaries would be in the hands of an experienced medical man, who is just as ready to work gratuitously as the ordinary physician or surgeon at the great hospitals. In such a case, I doubt if an individual can treat more than one hundred cases a year. In London alone, there are 40,000 cases requiring treatment, so that there is plenty of room for about 400 doctors doing this sort of work in London alone. As soon as we look at the problem in this way, it becomes clear that the work

requires very extensive and thorough organisation. It is certain that special departments for this work will be established at all the hospitals, but they must be distinct and independent like the special departments of hospitals.

But apart from hospitals, it will be necessary to establish numerous independent tuberculin dispensaries in every poor and populous district, so that the means of treatment may be ready at hand. We may suppose that in every community of 50,000 persons, there are about 200 or 300 victims of tuberculosis who cannot pay for proper treatment. Such a number could be provided for by one dispensary at which two doctors were employed who administered tuberculin treatment. Even if one paid each doctor a salary of £250 a year, the total cost of treatment need not exceed about £750 a year. Otherwise, as at present, the vast majority must suffer and die, because what administrative body, whether County Council, or Borough Council, or Board of Guardians, can provide treatment for 200 or 300 cases every year at a cost for maintenance alone, of about £5000 a year, which would be a *low* estimate of the cost of sanatorium treatment?

PART VIII

MR. LLOYD GEORGE'S INSURANCE BILL IN
RELATION TO THE TREATMENT OF CON-
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THIS Insurance Bill is framed after the model of the Imperial Act of Germany, and inasmuch as the German Act is specially devoted to the problem of dealing with consumption among the insured, and this problem forms one of the greatest and most expensive features of the scheme, it is worth while considering how far it is wise to copy an Act which was framed many years ago, when it was imagined that sanatoria offered the best means of realising the objects of the Act. Since this Act became the law of Germany experience has shown that sanatoria at their best are an unwieldy, expensive, and largely ineffective means of dealing with this great national problem. It is well, therefore, to review the system and its results with our eyes open, and in the light of actual experience.

If the German method has failed to realise the high expectations promised by the too enthusiastic advocates of sanatorium methods, and later experience offers a solution which is simpler, more economical, more effective, and free from the obvious disadvantages of a system which enforces loss of occupation and wage, and at the same time absolute idleness during many months, it would surely be an act of servile imitation and unwisdom to adopt a system merely because it formed part of the German Act of fifteen or twenty years ago. In considering this problem we must at the outset remember that this national scheme applies exclusively to wage-earners of £150 a year or less. We must disabuse our minds of the idea that we are considering the value of sanatoria for those who are able to devote money

and years to the treatment of this common disease, and focus our attention solely upon the scheme as it applies to those who cannot afford to sacrifice their occupation or their earnings, unless it can be shown that no other system can help them. Further, it must be admitted by all who are conversant with the purpose and working of the German Act, that the system aims not so much at the cure of the disease as the maintenance of ability to work for a limited number of years. The German Act is so far an economic success, because with an outlay of £20 or £30 a year it is quite possible to maintain the industrial efficiency of the human units for such a length of time (say two or three years) that in default of any other system a distinct economic success may be achieved. The only question that remains is whether this system as it has been exploited in Germany leaves nothing to be desired. In spite of the sanguine anticipations of the enthusiastic supporters of the Volkssanatoria of Germany, it is stated by high authorities that this system has so far not produced any appreciable diminution of the death-rate from consumption in Germany, nor is this a matter for surprise. This colossal machinery in Germany, involving an expenditure of several millions a year, has been able to deal with little more than 10 per cent of the multitude of sufferers, and there is striking evidence to show that the benefit to this small minority is, to a large extent, transient. Among the classes which would be affected by this legislation it is doubtful whether in the majority of cases the benefit would last more than four or five years. Accordingly, while we may speak of the economic success of this system because the beneficiaries themselves largely contribute to the support of it, we should be cruelly misleading the unfortunate victims if we allowed them to imagine that the system cured the disease in any large proportion of cases. The serious problem must be discussed from every point of view, and those who advocate the scheme propounded in Mr. Lloyd George's Bill must take care that upon the hustings rhetorical rhapsodies and picturesque generalisations do not deceive those who are called upon to give a vote in favour of a scheme which literally is offering

a stone to those who are crying out for bread. I cannot believe that the members of any party, much less of the Labour party, would support the idea of establishing sanatoria throughout the country if they knew that the system cannot reach the multitude, and that even in the case of the minority who might be fortunate enough to receive the benefit of treatment in sanatoria the net result would be that these favoured ones would be able to remain at work for another three or four years by virtue of this treatment. Failing any other system we might still support this scheme for sanatorium treatment, although the cost is so great and the benefit relatively small.

It is extremely difficult to assess the value of sanatoria for the poor. In England it is not possible to obtain any relevant evidence from existing sanatoria, because until quite recently no sanatoria existed which dealt exclusively with the class of people which would come under the conditions of the Insurance Bill. Nothing but careful and laborious investigations and compilations, existing over many years, during which all the cases treated must be kept under strict and continuous observation, can throw a clear light upon this complex and difficult problem. Such investigations have been made in Germany by the Imperial authorities, and the results show that the lower the wage the less chance the victim has of getting the better of this disease. Certain statistics of the Taxation Bureau of Hamburg show that the incidence of the disease is two or three times as great among those who have less than £100 a year as it is among those who have an income of over £100 a year. Now, if this incidence of the disease is greater in this class of society, it is a fair assumption that their ordinary life conditions either favour infection or lower their power of resistance. Accordingly, when they have been successfully treated, and are compelled to return to the ordinary conditions of their life and home, they are in a similar ratio likely to relapse. And where does Mr. Lloyd George's Bill make any provision for cases of relapse? Whatever sanatorium treatment may do, it does not establish any permanent immunity from the disease, and this is one of the weakest

features of sanatorium methods. Further, in these classes sanatorium treatment can only be carried out for a certain number of months, during which the patient is practically idle, and it is a severe strain upon the morals of these victims to be thrust into this state of idleness for many months, and then to return to the arduous labours of their everyday life. No doubt with graduated labour during treatment the demoralising effect of inactivity is diminished, but unfortunately this system of graduated labour cannot be applied indiscriminately, and hitherto its value has been proved in but a small and specially selected class of cases. This disadvantage has long been recognised, and the fact that "after-care" societies have sprung up as the inevitable corollary of the sanatorium system shows that the sanatorium dismisses its patients into the world in no sense fit to resume their ordinary duties. The very existence of these so-called "after-care" societies and farm colonies for those who have already enjoyed the benefit of sanatorium treatment is in itself a somewhat eloquent commentary upon the real value of sanatorium measures. I have seen such instances in which able-bodied men have gradually drifted into a state of invalidism as a result of this artificial, unnatural, and pampering method of treating the disease.

After all, is it worth while to establish and perpetuate a system of treatment which encourages a man to shirk his duties and live upon the charity of his neighbours? Such a system inevitably encourages unemployment, and at the same time breeds discontent and selfishness. For these reasons I have long since ceased to place individuals under these temptations, and have attempted to evolve a system which, without any of these disadvantages and disabilities, aims at interfering as little as possible with the life duties of the individual, and nevertheless restores him to health more rapidly and more effectively than a few months in a sanatorium can ever hope to do. These views, confirmed after mature consideration, compelled me to look beyond sanatoria, and I therefore lost no time in calling attention in the *Morning Post* to the respective merits of tuberculin dispensaries and sanatoria in the solution of this problem.

I wrote in the *Morning Post* of May 15th as follows:—

“While it will always stand to the credit of the Chancellor of the Exchequer that he should have forced the problem of dealing with tuberculosis among the poor upon the attention of the Imperial Parliament, it is to be regretted that he has mainly based his own scheme of relief upon a system which for the last fifteen years has been well exploited in Germany and has proved to be anything but a great success. Speaking from a tolerably intimate acquaintance with the purpose of the German sanatoria for the poor and their results, I cannot imagine the source of information which has prompted the statement that ‘the results are amazing in the number of cures that are effected.’ The results have unfortunately been so disappointing that for some years authorities have studiously avoided the use of the word ‘cure’ and merely speak of capacity or fitness for work (*Arbeitsfähigkeit*) as the test of their system. If the Chancellor of the Exchequer deliberately selects sanatoria as the pre-eminent means of treating consumption among the poor, he is offering a stone to people who are asking for bread. Germany has established a colossal system of insurance for its working people, and incidentally the health and life of the fighting unit is protected by this magnificent system of national insurance. It would be well for Mr. Lloyd George to supply the facts and statistics which have justified him in speaking of the ‘amazing number of cures.’ The Germans themselves have instituted searching inquiries in order to test the permanence of the results achieved by sanatorium methods in the treatment of tuberculosis. The careful statistics of Englemann (*Tuberkulose Arbeiten K.K. Gesundheitsamt*, p. 158) deal with the after-examinations of many thousand cases treated in thirty-one different sanatoria. ‘Of the cases discharged from sanatoria, not quite four-fifths were still fit to work six months after their discharge. At the end of three and a half to four years, on the other hand, four-fifths were either dead or invalids.’ ‘If one considers the number of deaths only, 25 per cent. were dead in a year and a half, while considerably more than half were dead three years after they were discharged from the sanatorium.’

"Dr. Ransome, a few years ago, after an examination of sanatorium results in Germany, said that the net result seemed to be that three years after treatment one-third of the cases were still able to work. Even in 474 cases in the early stage treatment in sanatoria, Englemann found that although in 66·3 per cent. no tubercle bacilli were found in the sputum, only 14·4 per cent. were able to work at the end of four years. In Hamel's statistics (*Tuberkulose Arbeiten K.K. Gesundheitsamt, 1904, Heft 2*) the records of 2685 patients are given. 77 per cent. on their discharge regained ability to work; 42 per cent. showed marked improvement; 20 per cent. improvement, but clinical cure was obtained in 5·3 per cent. and complete cure in 1·1 per cent. We must be thankful for very small mercies if we dignify these results as 'amazing' successes. We may estimate that in Germany at a cost of about one million a year 30,000 patients out of a suffering multitude of about 300,000 are treated every year in sanatoria, and the net result is that at the end of four years less than 15,000 out of this 30,000 are still alive; but even with such a scheme 270,000 sufferers are beyond the pale of grace. Yet someone has persuaded the Chancellor of the Exchequer that Germany has obtained 'amazing' results by means of sanatoria.

"Is it worth while to imitate such a costly, inadequate, and ineffective scheme when German authorities themselves furnish proof that the scheme hardly benefits 5 per cent. of the sufferers? No doubt from the State Insurance point of view and in an actual sense the system is a success, because at a cost of £30 or 30, half of which is contributed by the class receiving the benefit, the artisan or workman is able to continue at his work and earn his wages for two or three years even while he is suffering from the effects of the disease. I wonder whether Mr. Lloyd George would dare to explain to the beneficiaries of the system the real meaning of an 'economic cure'." But the most convincing evidence of the failure of sanatoria as a means of helping the poor already enmeshed in the coils of this disease is furnished by the after-examinations carried out by the pioneer of the Volks-sanatoria of Germany, Dr. Weicker."

(For further illustrations see pp. 215-217 of this volume.)

I then proceed:—

"I should not have been so cruel as to undermine the public faith in sanatoria if I had not convinced myself by careful and laborious observation that, although sanatoria fail to help the poor in their extremity, methods have been evolved by the master mind of Professor Koch which far transcend in their simplicity and real and lasting value all the methods which have hitherto been used. In order to test the value of the specific treatment of tuberculosis by means of tuberculin certain stringent and even exacting conditions must be imposed and observed. Firstly, the remedy should be the only remedy used in treatment. Secondly, the remedy should be exploited in a consecutive series of cases of all kinds (not merely specially selected cases) and all the cases fully treated should be published. Thirdly, after treatment the cases should be carefully watched and examined for at least three or four years before a final judgment is given upon the value of the remedy. Fourthly, the results should be arranged in three groups, or better, as in my own records, in five groups, according to the character and degree of the changes in the lungs. These conditions, I admit, disqualify the work of almost all observers, because, except by myself, tuberculin has never been tested under these conditions. My own observations have been carried out, as far as possible, under these conditions, and, therefore, should furnish trustworthy evidence of the value of this method.

"When I established my tuberculin dispensary in London I set out with the fixed idea of proving that in this huge Metropolis the poor afflicted with consumption, even in a relatively advanced stage, can be treated successfully by the use of tuberculin alone, without sacrificing either their employment or their wage. The patients continue to live under their ordinary home conditions. This disease affects so many of the poor of cities that it is literally impossible to deal in any comprehensive and effective way with the majority of the cases unless we can treat them while they remain in their own homes. The tuberculin dispensary alone offers a reasonable solution of this gigantic problem,

and I believe I am already justified in saying that this plan of dealing with the disease among the poor is a feasible one. Already the tuberculin dispensary at Kennington Road has been at work for more than a year, and during that time tuberculin has been consistently and continuously used, both for diagnosis and treatment, in practically all cases without any selection, except in the few cases which were obviously on the verge of the grave. Of the cases rejected, five out of seven died within, at most, four months of their being rejected. Apart from these absolutely hopeless cases there is only one case, a case of severe and persistent 'mixed infection,' which even after seven months' open-air treatment is still unfit for specific treatment. Many cases have been sent to the dispensary by doctors in the neighbourhood and elsewhere; accordingly, we are dealing with cases in which diagnosis is only too evident, and yet we have obtained successful results that failed to appear until tuberculin was used. A careful examination into the results of the work at the tuberculin dispensary has induced the Portsmouth Council, under the skilful guidance of Dr. Mearns Fraser, to adopt this method of treatment by means of tuberculin dispensaries before all others. This action of the Portsmouth Council, due entirely to the inspiration of Dr. Fraser, is the first instance in which a Municipal Council has deliberately adopted the tuberculin dispensary as the best means of dealing with tuberculosis among the poor. More recently the Inverness Council has adopted my scheme, and their Medical Officer, Dr. Mitchell, studied my methods at the tuberculin dispensary and has already inaugurated the system at Inverness. Then in Ireland at the present time there are two centres—one in Dublin and one in Naas, which has lately been inaugurated by the Countess of Mayo. During the last year scores of medical men have availed themselves of the opportunity of watching this work at the tuberculin dispensary in London, and I think I am justified in saying that 95 per cent of them have gone away with the conviction that the treatment is quite free from danger, not very easy to learn in a few visits, and extraordinarily effective if used with care, discrimination, and judgment.

* The results certainly show that in proper hands the treatment is productive only of good. I think therefore that the time has come for this particular mode of dealing with the problem to be honestly and openly considered, certainly before a single penny is spent for the purpose of building sanatoria. I am ready to admit that sanatoria also have a value, but sanatoria should be a mere supplement to tuberculin dispensaries. Tuberculin dispensaries alone can deal with the vast majority of cases of tuberculosis among the poor, at no greater cost than will provide sanatorium treatment for 5 per cent. of the sufferers. At any rate, before the Imperial Parliament pronounces definitely in favour of Mr. Lloyd George's chain of sanatoria throughout England and Wales and Ireland, it would surely be well to carefully and judiciously investigate the evidence which can be obtained directly at the tuberculin dispensary itself upon the merits or otherwise of this system. Mr. Lloyd George calculates that there are 300,000 to 400,000 victims needing treatment. Even if we had as complete a chain of sanatoria as they have in Germany at the present time, we could deal with no more than 30,000 out of this multitude, and all Mr. Lloyd George's funds for the purpose would be exhausted. Does Mr. Lloyd George propose, then, to establish a scheme which only benefits temporarily 30,000 cases out of 300,000 or 400,000? In the short space of a few years the benefits will have disappeared in more than half the cases dealt with, so that at this enormous expense hardly 5 per cent. of the sufferers are permanently benefited.

" Let it not be forgotten, too, that the sanatorium system compels the victim to give up his occupation and lose his wage. I quite admit that even with this great sacrifice, if the system restored the sufferer to health in the majority of cases, it might be economically sound to support it, but inasmuch as the most careful records show that when the poor have to return to their usual home conditions and surroundings the disease, after a short respite, reasserts itself and finally brings the victim to the grave, it is high time to look about for a better method. On the other hand, tuberculin dispensaries offer a form of treatment which in no way

obliges the sufferer to make any sacrifices. In the early stages of the disease, and sometimes in moderately advanced stages, the sufferer may continue at his work and earn at least something while he is being radically treated for the disease. Provided the work is not too strenuous and the hours are moderate it does the patient no harm to be thus occupied during treatment, though it may prolong the treatment by a few weeks. Secondly, the treatment is not costly; indeed, many a working man, say, earning two or three pounds a week, could afford to pay for the treatment himself. The cost of the remedy itself need not be more than a pound. There need be no costly buildings, so that the bulk of the money is really spent upon treatment. The cost of supporting the patient is also avoided, because he can live at home. This maintenance of the patient at sanatoria is a very large item in the expenditure; through the tuberculin dispensary this expense is entirely avoided. Quite apart from loss of wage, the cost of sanatorium treatment is at least twenty times as great as the cost of treatment at a tuberculin dispensary. This means that a sum of money sufficient to treat all the cases by tuberculin dispensaries, can, if applied to sanatorium methods, only deal with 5 per cent. of them. There is little doubt that if the money now devoted to sanatoria for the treatment of the poor of London were devoted to tuberculin dispensaries instead all the sufferers could be treated.

"The only further question that has to be settled is whether tuberculin dispensaries can do the work as efficiently as sanatoria. This is the crux of the question. If this can be proved discussion is at an end. It is upon this phase of the question that we need more light. In the first place, the question can only be settled by direct and positive evidence. In England unfortunately there is no evidence at all upon the question except the evidence which I and my pupils have furnished within the last year. Even in Germany and America the evidence is not quite satisfactory, because although the belief in tuberculin as the specific remedy for this disease is gaining ground every day, yet I know of no institution or place in which an effort has been made

to prove that tuberculin alone, without any sanatorium treatment at all, is in a large number of cases an efficient remedy for tuberculosis, including pulmonary tuberculosis. Ever since I have been working with tuberculin as a remedy I have taken care to exclude as far as possible all other factors to which some of the benefit or effects might have been attributed. Most Germans and Americans have used tuberculin as a mere adjunct in sanatoria. I have tried to show throughout by my work what tuberculin alone can do in checking the progress of tuberculosis and in eradicating it. I have used no other remedy or system, no sanatorium treatment, no drugs, such as creosote, which does harm rather than good, no open-air treatment, no over-feeding, no massage, no hydropathy. I have used tuberculin alone, and I venture to think that the results that I have obtained by this method are far better than the results of any other method.

"I hold therefore that at this critical juncture, when the Chancellor of the Exchequer proposes to spend some millions upon sanatoria and at least a million each year for maintenance, it is prudent to investigate a method which involves very little cost upon buildings and brings treatment within reach of the great majority of the sufferers at one-twentieth the cost proposed in his scheme for each individual treated, and with better individual results. Such an investigation need not be costly, because the evidence is ready at hand at the tuberculin dispensary in London, at Aldershot, and at the other tuberculin dispensaries. Here, at least, it can be shown that the system is simple, cheap, free from risk, and effective and popular among the patients themselves. The tuberculin dispensary is not what has been called a tuberculosis dispensary. The tuberculin dispensary depends for its success upon the use of tuberculin for diagnosis, for treatment, and for the subsequent control of the cases. A tuberculosis dispensary makes little of treatment at the dispensary, and is essentially a station for sending early cases to sanatoria. In my judgment, this is wrong. These early cases should be treated at the dispensary with tuberculin. Sanatoria should be reserved almost exclusively for cases of

mixed infections and complications. Neither the one nor the other is common in early pulmonary tuberculosis. Therefore early cases should not be sent to sanatoria, because they can be treated more simply, more economically, and better at a tuberculin dispensary. But my whole argument rests upon the assumption that tuberculin dispensaries can successfully deal with pulmonary tuberculosis. Once this is established tuberculin dispensaries, not sanatoria, will form extensive chains throughout the country, ramifying even into the smallest villages. It is for an independent, impartial, and competent commission to investigate this problem, which is the greatest problem in medical science."

THE FINANCIAL ASPECT OF THE PROBLEM

It is very remarkable how little serious attention has been paid to the financial aspect of Mr. Lloyd George's scheme, either by medical men or the advocates of sanatorium methods.

Since illustrations and arguments appeal the more strongly when they have a local colouring, let us carefully examine the problem of dealing with tuberculosis in London, so that we may estimate the character and extent, but especially the cost of the work that is to be done if we wish to bring within reach of the system the majority of sufferers.

It is certain that in London there are about 40,000 persons suffering from tuberculosis who cannot themselves pay for proper treatment.

Mr. Lloyd George's scheme would provide a dole by Act of Parliament amounting to £200,000 for buildings and £120,000 for the maintenance of sanatoria for dealing with 40,000 cases of the disease in London itself. For £200,000 about 800 beds would be provided. Since the average stay for each case should be at least six months, 1600 cases could be treated each year. Among the poorer classes more than 50 per cent. of the cases thus treated would relapse within three or four years, so that no more than 800 out of 40,000 cases would be permanently benefited. What provision does

Mr. Lloyd George intend to make for the 32,000 cases that are beyond the pale of grace?

Further, the maintenance of each case thus treated costs about £30. Accordingly the maintenance fund of £120,000 a year would support about 4000 cases out of 40,000. What sort of treatment are the remaining 36,000 cases to have when the fund is thus completely exhausted? Even if Mr. Lloyd George could by some sort of miracle make up the large deficiency of beds in the sanatoria so as to accommodate 4000 cases each year, he would still be dealing with only 10 per cent. of the sufferers. Unfortunately many cases derive little benefit from sanatorium methods, and many may need to stay in the sanatorium for a year before they receive any great benefit from this haphazard system of treatment. Apart from these immediate failures, we must bear in mind the numerous failures that develop a year or more after what is called successful treatment. These cases of immediate failure and virtual failure have had their chance, and Mr. Lloyd George's scheme can make no further provision for them.

Among the poorer classes of society the sequel to sanatorium treatment is little else than a long, wearisome chapter of heartrending relapses, ending in death in two or three or more years. Is it worth while to spend all this money to provide sanatorium treatment for a small fraction of the sufferers, when even they do not gain any great advantage by it? But what can be said in favour of a system which practically ignores and cannot possibly help the vast majority of sufferers?

If sanatoria were the best and only means of dealing with the problem, London alone would need, not 800, but at least 8000 beds for the work, and the cost of buildings would be £2,000,000, and the cost of maintenance would be about £500,000 a year. Even then in one year less than 50 per cent. of the cases could be treated. This is surely the *reductio ad absurdum* of sanatorium treatment for the poor—*tuberculi venter noster ridiculus ens*.

Now let us consider the financial aspect of tuberculin dispensaries.

I have proved that it is quite possible to treat the majority of persons suffering from tuberculosis in any form with tuberculin administered at simple dispensaries. The cost of building is a very small item, as it should be. In London we rent for £60 a year a house where two doctors can easily treat 300 odd cases every year. Thus London would need 100 such dispensaries for dealing with all the cases. Two doctors specially trained in the methods of tuberculin treatment would be required at each dispensary. Allowing £250 a year to each doctor and £300 a year for the cost of tuberculin, we should be able to treat 300, even 400 cases at a cost not exceeding £1000 a year. Thus the total cost of treating 40,000 cases in London needing assistance would be less than £100,000 a year.

Mr. Lloyd George is ready to spend £200,000 on buildings to deal with consumption among the poor in London. The interest on £200,000 at 4 per cent. would be £8000 a year. This interest would more than pay for all the buildings required by London to deal with tuberculosis by means of tuberculin dispensaries, and the capital would remain untouched. If the system proved to be a failure, the capital is not thrown away. So much for the buildings.

If Mr. Lloyd George is anxious to spend money on buildings, he will do so with far greater advantage by building special institutions for the reception of those advanced cases beyond the reach of any remedy, instead of building sanatoria which can never greatly help the poor. A system which removes one such individual to a place where he would be well treated, well fed, and well nursed would perhaps be the means of saving several lives. In Berlin it is stated that about 40 per cent. of the deaths from consumption occur in public institutions; in that way the health of the community is safeguarded. Strange that this practical method of preventing the spread of infection is approved by all medical authorities, and yet what medical authority has had the courage to put into practice this system which deserves and claims such universal approval? Money spent on such institutions is well spent, but the system would not win many political votes. Yet this is the first measure of prophy-

laxis that the Imperial Parliament should introduce in the interests of the victims of the community.

This work should be directly supported by the State, and should not be left as a burden to institutions supported by voluntary contributions. If the larger hospitals provide special wards for these cases, the Government should pay for the cost of the building and the maintenance of the patients. They are just as necessary in the interests of the community as asylums for the insane. No other institutions can deal with these cases, neither sanatoria nor dispensaries, so that for purposes of prevention such institutions are indispensable.

Mr. Lloyd George is ready to provide £120,000 a year for the treatment of the disease in London. £120,000 a year would more than cover the total expense of tuberculin dispensaries necessary to deal with all the cases of consumption among the poor of London.

I believe that I can prove before any proper tribunal by evidence that would be accepted in any court of law that tuberculin treatment yields far better results than sanatorium treatment, especially in the poorer classes; not merely better because the system can be applied at the same cost to twenty times as many individuals, but actually better individual results. Hence it appears to me that Mr. Lloyd George's scheme, based no doubt upon the statements and opinions of those who, without any personal experience, have trusted to and accepted the optimistic anticipations of twenty years ago, which have not been realised, can never really touch the fringe of the problem at issue, and not merely involves an enormous waste of public money, but will greatly disappoint the hopes of the few sufferers whom the system can actually reach. Literally the masses are crying out for bread, and Mr. Lloyd George is offering them a stone.

At best Mr. Lloyd George's scheme can benefit 5 per cent. of the suffering masses. What is to become of the 95 per cent. beyond the pale of grace? Surely, a dispassionate and earnest consideration of the financial obstacles in the way of the fulfilment of Mr. Lloyd George's optimistic generalisations should make the House of Commons pause before

it commits the country to such a wasteful and ineffective method of dealing with this national problem. On the other hand, if the money were devoted to tuberculin dispensaries the vast majority of sufferers could be treated, and treated with success, and in a large proportion of cases could earn their living while they were being treated. Think of the saving to the nation if the loss of occupation and earnings which is inevitable under Mr. Lloyd George's scheme were largely prevented! Surely it is wiser to help people to continue at work, if the work is not detrimental to health, rather than undermine their independence, encourage invalidism and idleness, and engender selfishness and discontent with their home and its surroundings by placing them for a brief space of time under the abnormal and even demoralising influence of life in a sanatorium.

With this expenditure too, and no more, nurses could be employed and specially trained to give instruction in the homes of the poor. In this way the risks of infection spreading in a family would be reduced to a minimum. In this work, too, health visitors might play a useful part. Still more important, Medical Officers of Health would be able to co-operate at every stage in the important administrative details which are indispensable in any sound system of prophylaxis. Thus there would be no overlapping of the duties of physicians, whose function it is to treat disease, and the duties of the Medical Officers of Health, whose special function is, *inter alia*, to prevent the spread of infection.

If tuberculin dispensaries were thus established as the essential part of the machinery to deal with the treatment of consumption among the poor in this simple, economical, direct, and effective manner, every penny of the public money would be spent in a way that would not be begrudged by the taxpayer. On the other hand, I myself, and many others, would resent the imposition of a system of taxation which we consider to be a mere waste of money, which cannot either gratify the taxpayer or greatly benefit the masses.

Lastly, if tuberculin dispensaries were established in London with this specific object, the services of about 200

doctors would be required to do the work efficiently. Some of these (leaders of the Profession) might be ready to offer their gratuitous services, but most of the workers at the dispensary should be paid. Allowing an average of £250 for each medical man, it would mean that £50,000 a year would be paid to the physicians who treat the disease; and in any system in which the treatment of disease is the essential function, it is both wise and just that the bulk of the money spent should go to those who do the all-important work. On the other hand, in Mr. Lloyd George's scheme, a sanatorium with 800 beds would hardly require twenty medical men; so that literally in carrying out the treatment at the sanatoria costing £50,000 a year, a mere pittance—£5,000 a year—would be paid for medical services. Surely it is clear, then, that if tuberculin dispensaries do actually achieve better results, self-interest alone should urge the members of the medical profession as a body to support this scheme, because the bulk of the money would be spent on direct, necessary, and invaluable medical services.

This system then can be recommended on medical, social, economic, and financial grounds. The system of tuberculin dispensaries considers the highest and best interests, firstly, of the victims of this disease; secondly, of the taxpayer; and lastly, even of the medical profession, upon whom alone the success or failure of any Bill insuring against sickness must primarily depend.

The system which employs in London alone 200 doctors to apply the most scientific and thorough methods of treatment to practically all the sufferers, including women and children, is surely better than the system which must exclude women and children and spends the same amount of money in treating by haphazard methods 5 to 10 per cent. of the sufferers. I trust, therefore, that before the Imperial Parliament makes a leap in the dark, in obedience to the blind and misguided enthusiasm of Mr. Lloyd George, in respect to this aspect (sanatoria) of the Insurance Bill, the merits of the system of tuberculin dispensaries may be carefully and accurately weighed in the balance.

Perhaps as the pioneer of tuberculin dispensaries for the

poor, I may take pride in the fact that in the short space of a year great interest has been awakened in the medical profession, and outside of it, in this new way of facing this great national problem. I owe more than I can express to the open-minded and public-spirited action of the *British Medical Journal* for the unstinted opportunities afforded me for explaining and discussing this system. The genial and generous editor, Dr. Dawson Williams, has not only helped and encouraged me in my work, but has also helped the medical profession to form a fair and deliberate judgment upon the essence of the scheme. Without this help I should have been but an "infant crying in the night," and the profession would be groping in the dark. The popular editor of *The Practitioner and Clinical Journal*, Dr. Eliot Cressy, who has throughout been my friend and colleague, has also done yeoman service in our cause.

Medical Officers of Health from every part of the country, from England, from Scotland, from Ireland and Wales, have visited the dispensary in London, and many of them have started, or are preparing to start, tuberculin dispensaries in their own towns and districts, after having first carefully and directly investigated the methods at head-quarters. At Aldershot Colonel Treherne has become an enthusiastic convert to the system, and many medical officers in the Navy have visited the dispensary and approved of the system.

Lastly, let me point out cogent reasons for the creation of tuberculin dispensaries as independent institutions. When I first came to London prominent men in the Health Department, entirely ignorant of the methods and system I was advocating, imagined that any treatment or method of treatment which was not carried out at the great London hospitals was hardly worth a moment's consideration. As a stranger, I was hardly in a position to combat this argument, yet I knew in my own heart that tuberculin was not used in these hospitals, either for diagnosis or treatment, in a proper way. I still hold that every large hospital will shortly have attached to it a special department, let them call it what they like, for this work, but it would be hopeless to imagine that hospitals alone can work the system. We must consider the circum-

stances of the poor, appointing convenient times for them and interfering as little as possible with their daily work. Further, we must win the sympathy and co-operation of the employers. I regret that several patients came to the dispensary and could not continue treatment because their employers would not allow them to leave work during working hours. This difficulty can be partly overcome if the patients can be treated in the evenings. Already at one of the branch dispensaries this system has been inaugurated and works splendidly, chiefly by reason of the self-sacrifice and devotion of the lady doctors who manage this dispensary; but even at hospitals this work, which is so exacting, and must be carried out with such care and precision, cannot be mixed up with any other work in an out-patient department. The dispensaries should be distributed in different parts of a city, so that the working-people shall lose as little time as possible.

The one ineradicable disadvantage of this system of treatment as of any other system is that it requires time, never less than six months, sometimes much longer, before a course of treatment can be completed. As a rule two visits a week are necessary, hence the importance of placing the dispensary to meet the convenience of the poorer classes. Not until the work at one of these dispensaries has been closely studied will it be clear that these conditions must obtain in order to ensure success. I think I may say that every medical man who has patiently studied the methods and purpose of the tuberculin dispensary will admit that I am merely asking for necessary conditions.

It is no idle statement that London alone needs at least 100 of these dispensaries if the work is to be done thoroughly, and the services of at least 200 doctors will be required. I should like to know how the hospitals in London could undertake this work; and yet when this objection was raised to my suggestion that tuberculin dispensaries should be independent of other institutions, it was hardly possible for me to meet the objection. All the better if the hospitals will undertake a small fraction of the work, but the great feature of the tuberculin dispensary system, as distinct from any

other system of dealing with tuberculosis, is that it makes a serious effort to bring satisfactory treatment within the reach, not of one sufferer in twenty, but of every poor man, woman, and child suffering from the disease.

I venture to think that if, and when, tuberculin dispensaries, not a few, but many, are scattered throughout the poorer districts in cities, they will have an enormous influence in disseminating knowledge about the disease, and the primary precautions necessary to prevent its spread, such as can be brought about in no other way. Not only will the people themselves recognise the value of treatment, but they will see the reasonableness of acting so as to cease to be a danger to their relatives and friends. In this way knowledge of a direct and useful kind will reach those whom it will most benefit more rapidly and more effectively than can be expected from the instruction of a few in a sanatorium, or the instruction by leaflets. Unfortunately the methods in vogue at the present time are superficial and act from a distance. I cannot myself suppose that even tuberculosis exhibitions can have any but a trifling, transient, and at best, remote effect upon those who chiefly need instruction.

One last word in explanation, and perhaps in self-defence. The whole of my argument rests upon the value of tuberculin in diagnosis, treatment, and prophylaxis. There can be but few persons in the whole of the United Kingdom who from their own experience are able to support my argument. Hitherto I do not consider that the use of tuberculin in diagnosis has been tested in any serious way, for the simple reason that the medical mind has been obsessed with the idea that its use for such purposes is fraught with danger, the imaginary danger that a test dose may light up a latent focus of disease. If this were true, I should have to reconsider my attitude and seek for some explanation why in twenty years' experience I have not once seen evidence to justify this idea of danger; but there is only one way of determining this point—the way of experience. Greater men than myself, with even greater experience, also say that the idea of danger is a foolish one. Most authorities who

have used tuberculin, as I have done, echo this view. Are we to stand with folded hands and listen to statements not based upon experience, but born in a timorous mind, untutored of experience and swayed by prejudice? I cannot, and I dare not, yield one jot to such a travesty of argument.

But while it is very easy to prove both the value and harmlessness of tuberculin as a diagnostic agent in early and doubtful cases, it is extremely difficult to form any safe, trustworthy, and comprehensive conception of the value of tuberculin in treatment and prophylaxis until one has used tuberculin in every reasonable way, in all kinds of tuberculosis, and in all stages of the disease; and, carefully and jealously watching the effects, not for a year or two, but for many years, and controlling the observations by every possible scientific and clinical test, can finally decide whether tuberculin used as the exclusive remedy can achieve results as good as or even better than the results of systems hitherto in vogue. This I claim to have done, and before I can admit any witness to the contrary he must be ready, not with mere statements, but with a series of accurate, prolonged, and careful observations, likewise controlled over a long period of time by clinical and scientific tests, which yield better results than those which I have obtained by means of tuberculin. This is real evidence, and I am ready to meet it; but I am not prepared to attach any importance or value either to general statements of personal experience, or, worse still, hearsay evidence or vicarious experience. I have never come across any evidence in favour of sanatorium methods for treatment of the poor which can compare with the evidence which I myself and many other pupils of Professor Koch's, can produce before any properly constituted tribunal, demonstrating the extreme value of tuberculin in arresting the early stages of the disease, and even converting more advanced stages into these early stages, so that not only the patient is benefited, but he ceases to be a danger to his neighbours.

It is only for me to state that by means of tuberculin early cases can be permanently arrested, while the majority

of cases in the second stage can be made to heal. If this can be done, and medical men will use tuberculin promptly and boldly to achieve these results, not in a few, but in the majority of sufferers, we should gradually but surely diminish the number of cases in the later stages, which are the essential sources of tuberculosis in all its forms in the human race. As far as I can see from my own experience of the disease, the system of tuberculin dispensaries for the poor offers not only the best, but the only solution of this great national problem.

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